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COLL. SANCTÆ INDIVIDUÆ TRINITATIS REG. ELIZAB. JUXTA DUBLIN

Thomæ Fitzpatrick
propter insignes in Anatomia
progressus

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Secundi Ordinis
dedit Schola Medica

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Examinatione habita initio Termini
S.S. Trinitatis A.D. 1855
quod testor Robertus Harrison
Professor Anatomiæ & Physiologiæ

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Given to my by the widow
of Dr FitzPatrick & by me to the College
Norman Moore

Oct 31. 1911.

THE DISEASES OF THE LUNGS, HEART, AND AORTA.

A

PRACTICAL TREATISE

ON

THE DISEASES OF THE LUNGS, HEART, AND AORTA:

INCLUDING

THE PRINCIPLES OF PHYSICAL DIAGNOSIS.

BY

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CONSULTING PHYSICIAN TO THE HOSPITAL FOR CONSUMPTION.

. . . . "Who knows but that one may discover the works performed in the several offices and shops of a man's body, by the sounds they make, and thereby discover what instrument or engine is out of order?"

R. Hook, 1705.

SECOND EDITION, REVISED AND MUCH ENLARGED.

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TO

SIR JOHN FORBES, M.D. D.C.L. F.R.S.

AND

NEIL ARNOTT, M.D. F.R.S.

IN

ADMIRATION OF THEIR EMINENT PROFESSIONAL CHARACTER,

AND

IN GRATITUDE FOR FRIENDSHIP AS DISINTERESTED

AS UNCHANGING.

ADVERTISEMENT TO THE SECOND EDITION.

The present edition has been carefully revised and much enlarged. Descriptions of several diseases, previously omitted, are now introduced; the prognosis of each affection is more carefully considered; the sections on diagnosis and treatment are materially extended; the theory of various acoustic phenomena has been examined afresh; and, in compliance with a desire expressed by many persons, such outline of the anatomical characters of each morbid state, as may answer the purposes of the practical physician, has been added.

Much as the Author would have desired this, it has been found impracticable, without inconveniently increasing the bulk, and altering the character of the volume, to print the details of cases in support of the statements and opinions tendered. His regrets on this score are however lessened by his being enabled to refer to the original narratives in his case-books at University College Hospital—records which possess at least the signal advantage of having been set down at the bed-side at the moment of observation.

PREFACE TO THE FIRST EDITION.

In the first of the two Parts, into which this work is divided, an attempt is made to describe the facts and principles of Physical Diagnosis in their applications to the Lungs, Heart, and Great Vessels. Although in the main confining himself to a description of physical signs, the Author has occasionally stepped aside to consider their mechanism,—yet not without the full consciousness, that in the present state of thoracic acoustics, and, indeed, of acoustics in general, all efforts of the kind must be merely tentative. If, on the one hand, the theories of the immortal discoverer of auscultation do not, in all instances, bear the test of clinical scrutiny, those of his German critics seem neither to be always in harmony with the principles of acoustics, nor accordant with the results of actual experiment. Whether such modifications as the Author has ventured to suggest in the theory of certain auscultatory signs, be of more satisfactory character, the verdict of his professional brethren can alone decide.

In the second Part, the symptoms, physical signs, diagnosis, and treatment of the chief diseases of the Lungs, Heart, and Aorta, are clinically described. The Author's design being to furnish, within a moderate compass, a guide to the detection and treatment of those diseases, he has systematically avoided, unless where some particular object obliged him to deviate from his plan, all inquiries into their general or special pathology.

When morbid anatomy was first seriously cultivated, the effects on medical practice were deeply depressing. It was naturally felt that the anatomical cure of the textural changes the scalpel revealed, was an impossibility. But a reaction has fortunately taken place; the conviction has gradually been forced upon observers, that many diseases texturally incurable, are mitigable by treatment to such a degree in their local and constitutional ill effects, as to be rendered comparatively innocuous. And of no diseases is this more true than of the chronic pulmonary and cardiac classes. Singularly happy as, at the time he wrote, appeared the motto chosen by Corvisart for his celebrated work on Diseases of the Heart,—"hæret lateri *lethalis* arundo;" a phrase so hopeless is at the present hour infinitely less appropriate.

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ERRATUM.

Page 631, line 26, *for* "See Cloetta," *read* "Lee, Cloetta."

PART I.

PHYSICAL EXAMINATION OF THE LUNGS, HEART, AND GREAT VESSELS.

INTRODUCTION—CLINICAL TOPOGRAPHY OF THE CHEST.

1. The existence of disease involves that of anatomical change, not only in the part originally and chiefly affected, but also in the structures immediately adjoining. There are a few apparently pure dynamic diseases, forming doubtful exceptions to this proposition; but, admitting their reality, they are not of sufficiently great importance to affect the general truth.

2. The anatomical changes thus arising may or may not be capable of accurate discrimination during life. When they can be so discriminated, experience has shown that their detection is not so much accomplished by means of the vital functional derangements of the organs implicated, as by the aid of various alterations in the physical properties of those organs,—as, for example, their density, their faculty of generating and of conducting sound, &c. So invariably do these alterations bear a certain and fixed relation to the physical nature of the anatomical conditions with which they are associated, that the discovery of the former is conclusive as to the existence of the latter. And not only the physical nature, but the precise limits

and the precise degree, of these conditions are disclosed by the alterations referred to,—which, for these reasons, constitute their *Physical Signs*. Interpreted by the observer, and not by the patient,—incapable, except in the rarest instances, of being feigned, dissembled, or even modified at will,—estimable in degree and extent with almost mathematical precision,—susceptible of indefinite refinement,—physical signs, like the whole class of objective phenomena of disease, are of immeasureably greater diagnostic, and considerably greater general clinical value than its subjective symptoms. Physical signs are, in fact, the true indices of the physical nature, extent, and degree of textural changes, and may be regarded as instruments of pursuing morbid anatomy on the living body. But just as their significance is sure and precise, so is the difficulty of mastering their theory and practice positive and great; and hence it is that Physical Diagnosis has gradually acquired for itself the importance of a special art.

3. The means by which physical signs are discovered or elicited, are called *Physical Methods of Diagnosis;* and these methods vary with the textural properties, functional attributes, and peculiarities of site of the organs examined. The diseases of the organs of respiration and of circulation are those of which the physical *signs* are the most varied, most significant, best understood, and most readily ascertained. The *methods* employed for their detection are:—

I. INSPECTION; II. APPLICATION OF THE HAND; III. MENSURATION; IV. PERCUSSION; V. AUSCULTATION; VI. SUCCUSSION.

These methods are, as nearly as is possible, applied to the parts themselves of which we desire to ascertain the condition,—to the external surface corresponding to them, when inapplicable to themselves. But the absence or presence of disease in the different thoracic organs, and, if it exist, its nature may sometimes be indirectly inferred by employing these methods in—

VII. The Determination of the Situation of Contiguous Parts and Organs,—which may consequently be considered an additional method of physical diagnosis.

4. All these methods agree in the general character of their direct and indirect objects. The *direct object*, the physician has in view with all, is the just appreciation of the sensations they furnish, and these are nothing more than the physical signs already spoken of; the *indirect object*, the reference of these signs to the physical states upon which they depend.

But a deeper object, to which these are merely preparatory, remains behind,—the determination of the pathological nature of those physical states. For this, familiarity with the laws of localisation, alliance and progress of thoracic diseases is indispensable. Physical signs, in truth, reveal physical conditions alone; they give no direct insight into pathological nature.

5. With the view of localising physical signs as precisely as possible, the surface of the chest has been divided into artificial regions; but as the assignment of limits to these regions is altogether arbitrary, it is not to be wondered that the boundaries adopted by different writers vary. Simplicity, as far as is compatible with the main object, should be especially aimed at in all such topographical arrangements; yet it scarcely appears possible, without a sacrifice of utility, to make the number of divisions less than in the following plan. They are designed to correspond, as far as is attainable, with important internal regions or outlines.

6. The chest is divided into anterior, lateral, and posterior regions. The *anterior* are called: supra-clavicular, clavicular, infra-clavicular, mammary, infra-mammary, supra-sternal, upper sternal, and lower sternal. The *lateral* regions are: the axillary, and infra-axillary. The *posterior* group comprises: the upper scapular, the lower scapular, the infra-scapular, and the inter-scapular. Of these regions the supra, upper and lower sternal are single; all the rest are double.

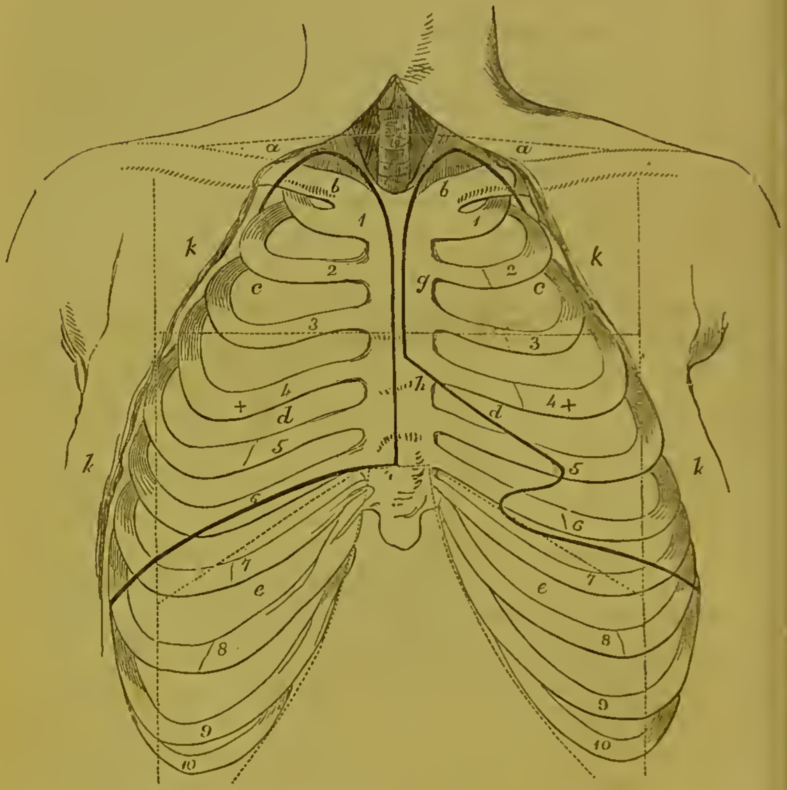

Diagram exhibiting the anterior regions of the chest, with their relationship to the ribs, and also the position of the edges of the lungs in calm (phrenic) inspiration. 1 to 10 inclusive, ribs; *a*, supra-clavicular region; *b*, clavicular region; *c*, infra-clavicular; *d*, mammary; *e*, infra-mammary; *f*, supra-sternal; *g*, upper sternal; *h*, lower sternal; *i*, trachea; *k*, integuments. The dotted lines indicate the boundaries of the various regions; the thick lines correspond to the outlines of the lungs, which rise to a maximum height of one inch and a quarter above the clavicle; (at the apex the *outer* border is carried to the line of *full* inspiration); + +, the nipples.—*Taken from an adult male, the trachea being tied after moderate insufflation of the lungs.*

7. The boundaries of these regions, and the more important structures and portions of organs corresponding to them, either within the chest or on its confines, may be stated as follow:—

Supra-clavicular.—Above, a line drawn from the outer part of the clavicle to the upper rings of the trachea; below, the clavicle; inside, the edge of the trachea. Here is found the triangular apex of the lung, reaching on the right side very slightly higher than on the left, with portions of the subclavian and carotid arteries, and of the subclavian and jugular veins; the first rib contributes to form a sort of floor for the region.

Clavicular.—This region comprises the portion of the clavicle, behind which lung lies, or, as nearly as possible, the inner half of the bone. Beneath the bone lies on both sides lung-substance; on the right side, at the sternal articulation, the arteria innominata just reaches the inner confines of the region, while the subclavian artery crosses it at its outer edge; on the left side the carotid and subclavian arteries lie deeply, almost at right angles with the bone.

Infra-Clavicular.—Above, the clavicle; below, the lower border of the third rib; outside, a line falling vertically from the "acromial angle" (formed by the clavicle and the head of the humerus); inside, the edge of the sternum. Within these limits are placed the upper lobe of the lung, on both sides; on the right side, close to the sternal border of the region, lie the superior cava, and a portion of the arch of the aorta; on the left, the edge of the pulmonary artery. On the left side, the inferior border of this region corresponds to the base of the heart. The bifurcation of the trachea taking place behind the arch of the aorta, on the level of the second rib, the main bronchus on either side is found in this region,—the right behind, the left a little below, the second costal cartilage.

Mammary.—Above, the lower border of the third rib; below, the sixth rib; outside, a vertical line continuous with the outer border of the infra-clavicular region; inside, the edge of the sternum. The contents of this region differ materially on the two sides. On the right side, the lung lies throughout immediately under the surface, extending downwards to the sixth rib, where the inferior border of the organ, turning off almost at right angles from the anterior, and gently sloping outwards, nearly corresponds to the lower edge of the region. The right wing of the diaphragm and the liver commonly rise to the fourth interspace. The fissure between the upper and middle lobes of the right lung passes obliquely upwards and backwards from about the fourth cartilage; that between the middle and lower, in the same direction from the fifth interspace. A portion of the right auricle and the upper and right angle of the right ventricle lie between the third and fifth ribs, close to the sternum. On the left side, the anterior edge of the lung passes obliquely downwards and outwards from about the level of the fourth cartilage, leaving a free space of variable size for the heart, and thus reaches the fifth rib; it then curves inwards and downwards to opposite the sixth rib or interspace, within the vertical line of the nipple,—whence it passes, at first, nearly horizontally, outwards. The anterior point of division of the lobes of this lung lies about the fifth interspace, below the nipple. The left auricle and left ventricle, with a small portion of the right ventricle about the apex, lie within this region.

Infra-Mammary.—Above, a line slanting outwards from the sixth cartilage; below, a curved line corresponding to the edges of the false ribs; outside, the outer edge of the mammary region prolonged; inside, the sternum at its inferior angle. On the right side the liver, with the lung encroaching to a variable extent at its upper part during full inspiration, occupies this region. On the left side lie the stomach and anterior edge of the spleen, which rises as high as the sixth rib; in the inner part of the region there is generally a portion of the left lobe of the liver, lying in front of the stomach.

Supra-Sternal.—A small region more or less hollow, bounded below by the notch of the sternum, and laterally by the sterno-mastoid muscles. The trachea fills it almost completely; it contains no lung; the innominate artery lies at its lower right angle; and in some persons the arch of the aorta reaches its lower border.

Upper-Sternal.—Corresponds to that portion of the sternum lying above the lower border of the third rib. Here are found the left, and a small portion of the right, innominate vein; the ascending and transverse portions of the arch of the aorta; the pulmonary artery, from its origin to its bifurcation; the aortic valves, near the lower border of the third left cartilage,—the pulmonary a little higher than these, and quite at the left edge of the sternum; and the trachea, with its bifurcation on the level of the second ribs. The inner edges of the lungs, coming slantingly downwards, almost join on the middle line opposite the same ribs, when the organs are fully expanded,—and lying immediately under the sternum, continue thus united as far as, and beyond, the lower edge of the region.

Lower-Sternal.—Corresponding to the remainder of the sternum, this region contains the main part of the right ventricle, and a small part of the left; the line of union of the heart and liver, with the diaphragm intervening; the edge of the right lung descending vertically along the middle line, and, at its upper part, a small portion of the left lung; and inferiorly, and deeply-seated, a portion of the liver, and sometimes of the stomach. The tricuspid and mitral valves, the latter somewhat posterior to the former, lie at mid-sternum opposite, or a little below, the upper edge of the region.

Axillary.—Extending from the point of the axilla above, to a line continuous with the lower border of the mammary region below, and in front from the posterior border of the infra-clavicular and mammary regions to the external edge of the scapula behind, this region corresponds to the upper lobes of the lungs, with, deeply-seated, the main bronchi.

Infra-Axillary.—Bounded above by the axillary region, anteriorly by the infra-mammary, posteriorly by the infra-scapular, and below by the edges of the false ribs, this region contains on both sides the lower edge of the lung sloping downwards from before to behind, with, on the right side, the liver, and, on the left, the spleen and stomach.

Upper Scapular and Lower Scapular.—Have the same boundaries as the fossæ of the scapula, and correspond to lung-substance.

Infra-Scapular.—Above, the inferior angle of the scapula and the seventh dorsal vertebra; below, the twelfth rib; outside, the posterior edge of the infra-axillary region; inside, the spine. Immediately underneath the surface, as far as the eleventh rib, lie the lungs; on the right side, the liver from the level of the rib just named, to the lower edge of the region; on the left, the intestines, occupying some of the inner part of the region, and the spleen of the outer. Close to the spine, on each side, but somewhat more on the left than the right, a small portion of the kidney encroaches on this region; and along its inner edge, on the former side, runs the descending aorta.

Inter-Scapular.—Occupying the space lying between the inner edge of the scapula and the spines of the dorsal vertebræ from the second to the sixth, this region contains on both sides lung-substance, the bifurcation of the trachea, the main bronchi, and the bronchial glands. It also encloses, on the left side, the œsophagus, and, from the third or fourth vertebra downwards, the descending aorta.

CHAPTER I.

PHYSICAL EXAMINATION OF THE LUNGS.

8. We propose here to give a general description of the various methods of physical diagnosis, and in the case of each method shall successively examine:—Its nature; its direct or immediate object; the manner of practising it; the conditions which are discovered by its means in the healthy state; such deviations from the ordinary standard of these conditions as are, nevertheless, compatible with health; and, lastly, the deviations from that standard, which are actually morbid and constitute signs of disease.

SECTION I.—INSPECTION.

9. By inspection of the chest, as a method of physical diagnosis, is understood simply the ocular examination of its external surface; by inspection are ascertained the conditions of exterior *form* and *size* of the cavity, and of the *movements* of its walls. The *form* of the chest is to be considered in respect of its *general configuration*, and the *shape of its various parts.* The *size* of the cavity is less important considered as a whole, than as composed of two divisions; the relative dimensions of these being the point of real consequence. The *movements* of the chest are *general* and *partial:* the *general* class includes those of *expansion* and of *elevation;* the *partial,* those of the *ribs in respect of each other*, and of the *intercostal planes.*

10. In order to insure correct results from inspection of the chest, the following precautions are to be observed :—the light must be good ; the surface fully exposed ; the patient's muscles relaxed, and all physical restraint removed ; and, above all, the plane on which he lies, stands, or sits, must be perfectly even. When the patient's state allows the observer the choice of the three postures just mentioned, the sitting ought generally to be selected. Inspection should be practised anteriorly, posteriorly, laterally, and from above downwards,—in the latter direction particularly, as a means of roughly ascertaining the antero-posterior diameter of the chest. Under all circumstances, it is of the last importance that the two sides, both generally and in their various corresponding parts, be closely *compared.* This observation applies with the same force to all other methods of physical examination ; without *comparison* of corresponding regions the utility of this kind of investigation would be very materially diminished. But in order that such comparison shall not be fallacious, it is essential that the observer should be fully alive to the numerous physical differences which naturally exist in corresponding parts of the two sides.

11. A. *Form.*—I. *In Health.*—The form of the chest of persons who have never had any affection of the thorax itself or its contents may be *regular*, or more or less *irregular.*

12. The adult male chest, if *regularly* formed, resembles, when viewed anteriorly, and exclusive of everything except its own immediate integuments, a cone having the narrow end uppermost ; its transverse diameter obviously exceeding the antero-posterior ; its two sides symmetrical, both generally and in their different parts ; the supra-clavicular spaces very slightly depressed ; the lower sternal region hollowed out in proportion to the stoutness of the individual ; the infra-clavicular regions gently convex ; the costal angles, formed by the union of the false ribs and middle line anteriorly, very nearly equal,—that on the right side being slightly the more obtuse ; the intercostal spaces visibly hollow in inspiration and expiration, unless the individual

be at all full in person; the lateral surfaces of the chest equally distant from the median plane,—as likewise the nipples, which are on the same level, that of the fourth rib or fourth intercostal space; and the different regions of the chest, considered in themselves, regularly shaped. Posteriorly, the shoulders lie on the same level; the spine is either perfectly straight, or inclines very slightly to the right at mid-back; and the vertebral sulcus, moderately concave from above downwards, more or less deep according to the fatness or thinness of the individual.

13. But it is comparatively rare to find a chest having in all respects the characters now enumerated. Certain deviations of form, perfectly compatible with a healthy state, both of the thoracic organs, and of the body generally, are of extremely common occurrence. It would follow, indeed, from the investigations of M. Woillez,* that the *regularly* formed chest, just described, exists in scarcely more than twenty per cent. of adult males, taken indiscriminately. The irregularities or *heteromorphisms*,† which render the chest non-symmetrical, while they are perfectly compatible with health, are by this author termed *physiological;* the name *pathological* being applied to those that are the manifest results of disease.‡

14. It is obvious that the chief, almost the sole, clinical importance of these "physiological" departures from regular form consists in the chance of their being mistaken for alterations of shape dependent on disease. Their frequency indicates the necessity of acquaintance with them: in 197 cases there existed 251 such heteromorphisms; 144 of these occurring in 111 persons

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* Recherches Prat. sur l'Inspection et la Mensuration de la Poitrine. Paris, 1838.

† From ἕτερος other, and μορφή form.

‡ *Regularly* formed chests are more common before than after the age of thirty, and in persons who follow sedentary pursuits or trades requiring little muscular exertion, or who have never laboured under thoracic disease, than among persons in the converse conditions. The previous occurrence of such disease does not, of course, necessarily imply the existence of irregularity of form.

who had had thoracic disease, 107 in 86 individuals who had all their lives been perfectly free from such disease.

Physiological heteromorphisms may be congenital or acquired, and general or partial. The *general* are those in which the natural relations of the different diameters of the chest are altered; the *partial* consist of local defects of symmetry, exercising no influence on the general shape of the thorax.

15. Again, certain local irregularities of form may be either of physiological or of pathological origin: disease may produce in one chest precisely the same alteration of shape that accidental circumstances, in no wise impairing health, effect in another. When a deviation of form, which may be thus either morbid or not, presents itself, its mode of origin can only be positively determined by the absence or presence of other signs denoting subjacent disease, or by the previous history of the individual showing that he has or has not suffered from pectoral complaints.

16. II. *In Disease.*—Alterations of form and of position of the whole thoracic surface, or of its parts, if considered in regard of *their physical characters*, may be referred to the following species:—(α) Expansion and Bulging; (β) Retraction and Depression; (γ) Procidentia and Elevation; (δ) Curvature; (ε) Distortion.

17. (α) *Expansion* signifies a change of shape of the chest, in which one or both of its sides is generally prominent; *bulging*, a local or circumscribed expansion, the remainder of the thoracic surface being either in the natural state or affected with some other species of irregularity. Expansion of one side, produced by some force acting from within outwards (the elasticity of the lung having been first destroyed), is best seen in cases of abundant pleuritic effusion, with or without pneumonia; in pneumo-thorax, hydro-pneumo-thorax, and general vesicular emphysema; less clearly in hypertrophy of the lung, intra-thoracic tumors, and extensive hæmo-thorax. Simple pneumonia and hydro-thorax have not yet been proved to produce it; nor are any affections of the heart or great vessels capable, even as

matter of theory, of doing so. Expansion of either side is never a physiological heteromorphism.

Bulging occurs at either base in pleuritic effusion and in pleuro-pneumonia; in emphysema appears above and below the clavicles, and has been observed in those regions in cases of simple pneumonia of the apex; exhibits itself in various sites in cases of circumscribed pleurisy and intra-thoracic tumor; in the right infra-axillary region in cases of enlarged liver, and in the left of enlarged spleen; in the mammary and lower sternal regions in pericardial effusion and hypertrophy of the heart, and in the upper and central parts of the chest in cases of aortic aneurism. But on the other hand, bulging frequently occurs as a natural condition in the following positions: the right back inferiorly; the left front inferiorly, with or without twisting forwards of the free edges of the ribs; the upper sternal region; the second costal cartilages, either or both; and the left sterno-mammary regions. Such non-morbid bulgings simulate those produced by pericardial effusion, aneurism of the aorta, pleuritic effusion, &c.

18. (β) *Retraction* and *depression* are the converse states of expansion and bulging: the former, a general sinking of the walls on one side; the latter, a similar condition limited to one spot or region. *Retraction* never exists without reduction of size of the lung, produced either by extrinsic pressure or by changes in its own substance. Now, pressure is essentially concerned in cases of pleuritic effusion: the lung, reduced to a small bulk by the pressure of accumulated fluid, deprived of its elasticity, and bound down by exudation-matter, is unable to resume its original volume on the removal of the fluid by absorption,—the side consequently yields inwards under the weight of the atmosphere. The exudation-matter aids materially, through its characteristic force of contraction, in producing this result, by diminishing the bulk of the lung,—not, as might be supposed, by actually dragging the wall of the chest inwards. On the other hand, changes of the lung-substance, reducing its

bulk, occur in tuberculous disease, the absorptive period of pneumonia, in cancerous and certain exudative infiltrations of the lung, and in collapse and atrophy of the organ consequent on the inaction entailed by pressure on its main bronchus by enlarged glands, tumor, or aneurism. *Depression* attends the same morbid states, when more limited in extent and influence. In estimating the clinical value of depression, the observer must remember that it sometimes occurs in the lower sternal region, and, symmetrically, in the infra-mammary regions, independently of disease.

19. (γ) *Procidentia* is that state in which the position of a part is lower than natural; *elevation*, that in which it is higher. Examples of *procidentia* are seen in the lowered position of the shoulder, of the ribs laterally and of the nipple in chronic pleurisy with retraction. The shoulders are not always naturally on the same level, however; and the left nipple is, in healthy persons, frequently lower than the right. I once met with *elevation* of the shoulder, on the *same* side as retraction of the parietes from chronic pleurisy.

20. (δ) *Curvature* signifies that deviation of the various axes of a part, in which, notwithstanding, some degree of regularity of form is retained; *distortion*, a displacement of the same kind fundamentally, but one in which the deviations are so numerous and so considerable that all trace of regular shape is lost. The spine, sternum, clavicles, and ribs, are subject to the former of these displacements in connexion with disease of the subjacent organs. Thus the dorsal spine becomes laterally curved in cases of chronic pleurisy with retraction; the ribs, in extreme cases of the same disease, undergo such torsion on their longitudinal axes, that their upper edges become external; the sternum yields sidewards under the pressure of intra-thoracic tumors, and the clavicle twists downwards and inwards in some cases of tuberculous destruction of the apex of the lungs. Some slight deviation of the dorsal spine to the right, I have found to be more common than perfect straightness in male adults with

sound chests; and the sternum sometimes naturally inclines to one side.

Here may be included the peculiar conformation, called "pigeon-breast," characterised by flattening of the lateral regions, and prominence, with arching forwards, of the sternum. This is doubtless a congenital malformation in some cases; but it may also be an acquired deformity. As has been satisfactorily shown by Dupuytren* and Mr. Shaw,† this alteration of form may be produced, especially in the flexible chest of childhood, by protracted difficulty of breathing depending on obstruction in the upper air-passages. The principle of its production will be explained further on [31].

The pigeon-breast seriously damages the natural relationship of the heart and lungs, and modifies both the heart's impulse, and the mode of conduction of its sounds.

21. B. *Size.*—No practically useful rule can be laid down respecting the ratio of the natural visible dimensions of the thorax to those of the body generally: the proportion varies widely in different individuals enjoying robust health.

22. There is no *visible* inequality of size in the two sides of a well-formed thorax. Numerous deviations from the natural relative dimensions of the different parts of the thorax occur in consequence of disease; but as they are always to be more accurately estimated by measurement than by inspection, and in some cases only to be ascertained at all by means of the former, the consideration of their different varieties is deferred to the section on MENSURATION.

23. C. *Movements.* I. *In health.*—(*a*) The *general* movements, or those in which the entire thorax is concerned, are of *expansion* and of *elevation.* In health these two kinds of movement are so intimately associated and agree so closely in proportional amount, that it is unnecessary to consider them separately; in certain states of disease they are very differently affected.

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* Mémoires de Chirurgie.

† Medical Gazette, 1842.

24. During *inspiration* the walls of the chest diverge from their central axis,—the sternum and the anterior segment of the ribs passing somewhat forwards, the lateral outwards, and the posterior backwards, from that axis (*expansion-movement*). At the same time the anterior walls, and, with the exception of the three or four last ribs which are on the contrary depressed, the lateral walls also, rise upwards (*elevation-movement*). Both these classes of movement are, in the majority of people, slightly more active at the middle than the commencement or close of inspiration, but free from all jerking inequality of rhythm. In ordinary breathing these movements are in the direct ratio of the antero-posterior and transverse enlargement of the lungs,—but not precisely so of their vertical enlargement, in as much as there is no constant proportion between costal and diaphragmatic movement. The rapidity, the energy, and the extent of the expansion and elevation-movements, bear a direct proportion to each other under all circumstances of health, unless volition interfere to pervert the natural order of things. On the other hand, the absolute amount of all three characters varies within sufficiently wide limits in different persons,—but is found to increase, as a rule, in the direct ratio of the easy mobility of the frame-work of the chest (hence greater in youth than age), and the height of the individual.

25. During *expiration* the walls of the chest are restored to their previous condition by the converse movements of *retraction* and *depression*,—with greatest rapidity during the middle part of the act.

26. In each act or respiration the motions of expiration follow those of inspiration so closely, that no distinct pause is perceptible between them; when expansion and elevation cease, retraction and depression appear to begin, precisely as the audible pulmonary sounds by which they are accompanied.

27. If the entire time occupied by a respiratory act (that is, from the beginning of one inspiration to the beginning of the next) be represented by 10, the value of the duration of the

inspiratory movement of the costal walls may be estimated approximatively at 5, of the expiratory at 4, and of the pause between the expiratory and succeeding inspiratory movement at 1: the period of thoracic motion is to that of rest, as 9 : 1. The movement at the close of expiration is so small in amount, and so slowly effected, that it is very difficult to fix accurately the instant at which the actual rest, dividing any two succeeding respiratory acts, commences—hence, doubtless, the difference in the above ratio, which I have been led to adopt as the mean result of numerous observations, from that set down by some other writers.

28. In health the *extent* and *frequency of repetition* of the movements of the thorax are in the direct ratio of the *duration* and *intensity* of the pulmonary respiration-sounds.

29. (*b*) The *partial* movements of the ribs on each other,—movements, practically speaking, limited to special situations,—are best appreciated by application of the hand.

In perfectly calm breathing the intercostal spaces continue visibly hollow, both during inspiration and expiration,—more so, during the former than the latter act. In the male this is most obvious in the infra-axillary regions; in the female in the infra-clavicular: in both sexes the fact can only be ascertained, where the individual is thin. In forced breathing the deepening of the interspaces is still more marked in inspiration; whereas during expiration they become more or less perfectly flat.

Further, the post-sternal hollow, and the supra-clavicular regions, seem to sink slightly inwards in calm inspiration, filling out again in expiration.

30. But the movements of the walls of the chest are not the only ones, dependent on respiration, which are of clinical importance. The enlargement of the lungs, and descent of the diaphragm in inspiration, forces down the subjacent viscera, and causes protrusion of the abdominal walls, especially anteriorly; during expiration these walls recede. Hence in *ordinary* or *calm* breathing, which is mainly effected by the descent of

the arch of the diaphragm, the amount of abdominal is greater than that of thoracic expansion-movement, and the former commences sensibly before the latter.

There is a striking difference, however, in this point of view, between the sexes; and the statement just made applies in point of fact to the male only. In the female the abdominal expansion is almost null, and always slightly posterior in time to the upper costal; neither do the lower ribs move notably; whereas the clavicles and infra-clavicular regions rise and fall with freedom. The adult male seems to the eye to breathe with the abdomen and the lower ribs from about the tenth to the sixth; the adult female with the upper third of the chest alone.*

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* The cause of this difference in the sexes is not satisfactorily determined. It is yet a point *sub judice*, whether, and to what proportional extent, the discrepancy of adult life prevails in infancy and early youth. I have examined a considerable number of female children, aged between four and ten years, who had never worn stays, or any substitute for these, and found in them the predominant infra-clavicular action of the adult. But the excess of upper movement is very positively less than among their seniors. On the other hand, Boerhaave (Prælect. Academ. § 623. tom. v. p. 144. Ed. Haller, Amst. 1744), one of the earliest observers of the difference in the respiratory action of the sexes, speaks as though the boy and girl of "one year old" breathe as distinctively, the one with the abdomen, the other with the chest, as the full grown man and woman. *Per contra*, Bean and Maissiat affirm, that in earliest infancy, and often up to the third year, the respiration is abdominal in the female as well as in the male. It has appeared to me positive, that in earliest youth, when the pectoral and ventral modes of breathing first become obvious, the chest action in the female is more *general*, and less limited to the upper regions, than at a later period. *Age*, then, does seem to me to exercise an influence upon, or to be connected with, the typical breathing of the sexes.

Social position has no modifying power; the washerwoman and the peeress breathe exactly alike.

The *habit of forced breathing* is not without modifying power on the calm action of both sexes. For instance, the extensive play of the upper regions in full chested *soprani*, kept up in the exercise of their art for many hours daily, ends by increasing the amount of infra-clavicular movement in ordinary conversational breathing. It has appeared to me that, even in

In *forced* breathing this sexual difference disappears; in both sexes the pectoral movement is out of all proportion greater

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tenor singers, some perversion of the ordinary condition—some degree of unnatural infra-clavicular movement—may be detected in calm respiration.

But what influence does *dress* exercise? Looking at an adult female, and remembering her habit of drawing in the lower ribs by apparatus more or less unyielding, the inference seems unavoidable that the reason why a woman does not breathe like a man is, that her mode of dress mechanically obstructs phrenic play. Certain mischiefs entailed by tight lacing we see positively in displacement of the liver;—in mis-shapement of it, so that its height is made to exceed more or less its breadth;—in alterations of its texture, so that true lobular substance is replaced to a greater or less depth by induration-matter functionally inert. We see them exhibited in displacement of the heart;—in narrowing of the lower intercostal spaces, &c. And if, from certain of the facts concerning age, just passed in review, we are forced to the admission, that the activity of infra-clavicular respiration-movement in the female is in the main designed by nature, and independent of extraneous influence, still I cannot help thinking that the great excess of that movement, and the limitation of breathing play to the upper thorax in the civilised adult female, are due in no small measure to the use of unyielding cases interfering with inferior costal and phrenic action. The agricultural female labourer, who knows not stays, breathes more like a male than the town female. Besides, during sleep, the conditions of pectoral and ventral action in the female are much less strikingly different from those in the male than in the waking state; the waist is relieved for a time from constriction. And further, the male and female dog breathe almost exactly alike, as do also the horse and mare; the action is abdominal and lower costal.

It would seem then that stays are in part productive of the peculiarity of adult female breathing, but certainly are not its sole cause. Boerhaave, and his commentator, Haller, however, holding that the sexual difference obtains from birth, looked upon the free upper costal action in the female as a pre-ordainment to meet the difficulties of pregnancy. "Nisi hanc," says Boerhaave, "in fœminâ diversitatem natura fecisset, gravidæ perpetuâ dyspnœâ laboravissent, æque ac viri hydropici." But it seems here to be forgotten, that if the illustration be sound, ascitic females ought to escape dyspnœa. The final cause of the difference in the sexes is of less interest, however, than the mechanism by which it is actually worked out; but of this also, nothing is known. Haller ascribes the predominant costal action in females to the greater flexibility of their bones and cartilages (Op. Cit. pp. 98, 145). The upper interspaces are relatively wider in the female, the lower in the male; but is this effect, or cause, or neither one nor the other?

than the abdominal; and even in the male the expansile action commences superiorly.

Various postures, which interfere with action in limited parts of the chest or abdomen, throw extra-work on others, and thus alter the mechanism of respiration. Thus, in calm breathing, the male, when lying on one side, exhibits extra-costo-abdominal movement on the other; in the supine posture there is no perceptible movement in the perinæum, in lateral decumbency sufficient rise and fall takes place there to count the respirations by.

31. *In Disease.*—(*a*) The general movements of simultaneous expansion and elevation are liable to *diminution*, either from instinctive avoidance of pain, paralysis of the muscles naturally producing them, or a material obstacle in the condition of the pleura or lung. Thus in pleurodynia, intercostal neuralgia, and at the onset of pleurisy, the first cause acts; in cerebral and in spinal paralysis, the second; in the course of pneumo-thorax, pleurisy, and pneumonia, of obstruction of the main bronchus, of gaseous, liquid or solid accumulations in the pleura, of consolidation and rarefaction of the lungs, the third. On the other hand these movements *exceed* the healthy standard, where a muscular effort is made to overcome some obstruction, seated *low* in the chest, as in spasmodic asthma, and especially if the diaphragm be mechanically interfered with, as in pericarditis with copious effusion. Or they may be *perverted:* if the *upper* air passages be obstructed, either from disease in themselves (œdema of the glottis; laryngitis, simple, œdematous, or croupal; tumors; foreign bodies, &c.) or in neighbouring parts, (enlarged tonsils; pharyngeal disease,* &c.), or from spasm of the glottis (as in epilepsy, hysteria,† laryngismus stridulus, pertussis and

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* Constable, U. C. H., Females, vol. ix. p. 97. Cancerous ulcerated opening between œsophagus and trachea. Two days before death, base of chest expanded, the apices sank in, in inspiration. Yet the apices proved sound, the bases congested.

† Spicer, U. C. H., Females, vol. ii. p. 147, Jan. 1847.

chorea), the chest, instead of expanding during inspiration, will actually retract, especially below and at the sides, while the abdomen enlarges, in proportion to the amount of obstruction. In many of these affections the rhythm of the movements becomes jerking and uneven. The more flexible and expansile any given chest is in health, the more marked will be this perversion, where obstruction exists above the bifurcation of the trachea: it attains its maximum consequently in childhood. The explanation is easy. The small quantity of air inspired makes naturally to the nearest air tubes, those of the upper lobes, while none reaches the lower; the inferior parts of the chest are consequently driven inwards by atmospheric pressure. This state of things becomes permanent where its cause is permanent; and thus is engendered in some cases the so-called pigeon-breast [20]. Instead of this bilateral perversion, the phenomenon will be unilateral, if the obstruction affect the main bronchus on one side only; or it will be still more limited if a bronchus of second or third calibre be alone affected. Now, whenever any cause seriously impairs the expansion-movement of one side only, the expansion of the other is liable to increase; and similar deficiency of action, limited to a part of one side, may be made up by excess on the rest of that side: the law is the same as for the audible phenomena of respiration. It holds good, too, where the obstruction is parietal, as in cancerous infiltration of the wall of either side of the thorax.

Want of power in the respiratory muscles, whether from debility or from paralysis, will impair the chest-motions; in the tetanic spasm, whether morbid or from strychnia, the walls are fixed.

32. (*b*) The relation of the movement of expansion to that of elevation may change completely; the former, for example, being almost totally suppressed, while the latter becomes even peculiarly obvious. When the lung-substance is more or less impermeable, either locally or generally, and either from disease within itself or pressure from without (as in cases of tubercle, pneumonia, pleurisy, pleuritic and pericardial adhesion, intra-

thoracic tumors or aneurism), this kind of perversion in movement will exist, either locally or generally, according to its cause. It is especially marked on forced inspiration; volition may drag the thorax upwards, but cannot expand impermeable texture. In vesicular emphysema, while the elevation-movements are carried to an extreme point, there may be no expansion at all, nay even slight retraction at the base, during inspiration.

33. (*c*) The *rhythm* of the *respiratory act* is likewise subject to change; the duration of the expiratory movement may become considerably greater than that of the inspiratory. This is observed whenever physical obstruction exists, in any part of the passages from the nares downwards, to the exit of air from the lungs; and also where, as in vesicular emphysema, the elasticity of the lung is destroyed. In the latter affection the expiratory movement may be two-and-a-half times as long as the inspiratory.

34. (*d*) The proportion naturally subsisting between the extent and frequency of the *movement of elevation* on the one hand, and the duration and intensity of the *respiration-sounds* on the other, may be altogether perverted: the former may be greatly increased in amount; while the latter have undergone remarkable diminution. This state of things constitutes one of the most remarkable features of diseases, where spasm affects the bronchial tubes or glottis, and obstructions of physical character exist in the larynx, trachea, or larger bronchi. The inspiratory action is abrupt and short, the expiratory prolonged.

35. (*e*) Again, the relationship of the thoracic and abdominal movements may change completely. Thus all conditions interfering, either dynamically or physically, with the movements of the diaphragm, while they impair these, give excess of energy to the thoracic class. Inflammation of the diaphragm, or of the serous membranes coating it, great fluid effusion in the pericardium, solid, fluid, and gaseous accumulations in the abdomen, pervert in this manner the natural order of things. On the other hand, the diaphragmatic movements may be increased by

certain irritations of the phrenic nerves, in pleurodynia, intercostal neuralgia, the painful periods of pleurisy, in diseases or injury of the spinal cord below the phrenic nerves,—and when compared with the amount of expansion-movement of the thorax, in obstructive diseases, functional or mechanical, of the air passages.

36. In the natural state the costal angles are nearly equal,—the left being slightly the more acute of the two. All affections, contracting either side of the chest to the exclusion of the other, increase the acuteness of the costal angle on the former,—as chronic tuberculisation, chronic pneumonia, chronic pleurisy with retracted side, &c. All affections augmenting the positive bulk of either lung widen the costal angle on the affected side,—as rapid and general hepatisation, and acute general tuberculous infiltration.* The more fully the base of the lung is implicated in the disease, the more obvious will be the effect. Where the costal angle is narrowed, its costal limb is depressed; where the former is widened, the latter is raised.

37. In estimating the thoracic movement of calm breathing in disease, the sex of the patient must always be borne in mind. An amount of infra-clavicular expansion, ample for a male, would be below par for a female: the converse is true of infra-axillary action. Various morbid states pervert the natural sexual condition,—giving the female the lower action of the male thorax, the male the upper mobility of the female thorax.

38. The intercostal spaces are widened by fluid and gaseous collections in the pleura, by intra-thoracic tumors and aneurism,—by certain kinds of enlargement of the heart, by hydropericarditis,—by extensive plastic infiltration of the lung, when sudden and in the acute state. Fluid in the pleura or pericardium may, in addition, cause bulging of the intercostal planes, irrespectively of the nature of the fluid, whether purulent,

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* Hodson, U. C. H., Males, vol. ix. p. 17. In this case of combined acute tuberculisation and pneumonia, while increase of bulk existed inferiorly, diminution by interstitial contraction had set in superiorly.

serous, or bloody. The conditions of bulging will be more fully considered with the history of Pleurisy.

The interspaces undergo narrowing in the absorption-period of pleurisy and pericarditis, and in all affections causing marked contraction of the lungs.

All movement of the intercostal planes is annulled in certain stages and conditions of pleuritic effusion and of intra-thoracic tumor. Their movements are liable also to perversion: the spaces may bulge beyond the level of the ribs during expiration; they may bulge too in inspiration: and this in different parts of the same chest at once tuberculous and emphysematous.

39. If fluid be accumulated in sufficient quantity in the pleural sac to cause marked bulging of the interspaces, an undulatory movement caused by respiration may sometimes be seen. If, in addition, the costal plane be perforated, and a fluid collection in the walls of the thorax communicate at all freely with another in the pleura, an inspiratory and expiratory fall and rise of the former is distinctly visible.

SECTION II.—APPLICATION OF THE HAND.

40. By application of the hand and palpation, are meant the acts of laying the hand on, and feeling, the external surface of the chest. The objects of these acts is to ascertain the *form* of the different regions of the thorax (little or no information can be derived from them regarding the *general* conformation of the cavity); the state of the *general*, and especially of the *partial*, *movements* of the walls; the amount of *vibration* communicated to the hand from those walls, and the existence or absence of *fluctuation* within the chest.

41. In employing this method of diagnosis, the palmar surface of the fingers and hand should be laid gently and evenly on the surface. If the object be to investigate the form or movements of the thorax, this is the only precaution, in addition to those recommended for the proper performance of inspection, which it is necessary to observe; if the thoracic vibration be

the subject of examination, it is advisable to place the patient in the horizontal posture.

42. Application of the hand is less useful than inspection in ascertaining the amount of *general movement*, taken as a whole, existing in any given thorax; but it is greatly more effectual in *locally* distinguishing *expansion* from *elevation*-movement, and in analysing the *partial* costal movements.

Thus in chronic pleurisy with retracted side a good deal of elevation-movement may be felt during inspiration, while the total absence of any action tending to fill out or expand the hand laid on the surface is readily ascertained. The same state of things may constantly be established in the infra-clavicular regions, when the apex of the lung is consolidated. In this case, and also in empyema, the thoracic walls, above and below the clavicle, may fall in during inspiration and expand during expiration, while the elevation-movement pursues its natural course and rhythm.

43. In the natural state, calm inspiration causes divergence of the lower ribs, and convergence (as originally shown by Haller, and recently by Dr. Sibson) of the upper; while an intermediate set, from the fourth to the seventh, undergoes scarcely any change. Expiration produces the converse effects. The amount of alteration is greatest superiorly in women, inferiorly in men; and in both sexes greater anteriorly and laterally than posteriorly. Forced breathing widens and narrows the interspaces proportionately more; the convergence of the infra-axillary ribs in the male during extreme expiration is particularly remarkable.

In the lower interspaces of the male, it is easy enough by placing the thumb or a finger on their surface to follow these movements. But it is very difficult to detect the inspiratory convergence in the upper spaces. When a finger is placed in an upper intercostal space, and the thinner the subject of the observation the better, it seems to be compressed by the adjacent ribs during expiration, and relieved of all pressure during inspiration—just, though not to the same amount, as in the lower interspaces. There is, however, some fallacy here; for I have found, by performing artificial respiration, after the removal of the integuments, on the dead male subject, that the upper ribs do actually converge during that movement. The amount of approximation, even in persons with wide intercostal spaces, appears to me not to exceed one-sixteenth or one-twelfth of an inch at the outer edge of the costal cartilages.*

Allied in mechanism to the expansion-movements, these diverging-movements may nevertheless be affected differently from the former in disease. Thus, in cases of chronic pleurisy, with contracted side, the elevation-movement may, during inspiration, be still perceptible in the infra-axillary region, and the expansion-movement be absolutely null, while the ribs actually converge. This inspiratory convergence of the ribs has appeared to me indicative of subjacent pulmonary consolidation and pleuritic *agglutination* combined.

44. *Vocal vibration, or fremitus.* If the hand be applied to the surface of the chest of a healthy individual, while speaking, a vibratile tremor is perceived by the fingers. This vibration, delicate under all circumstances and readily deadened by too forcible pressure of the hand, is, generally speaking, in the direct ratio of the graveness, coarseness of quality, and loudness of the speaking voice, and hence, as a rule, more marked in adults than in children, in males than in females. It is often altogether deficient, indeed, in females and children. *Cæteris paribus* it is more intense in long-chested than in short-chested persons; and markedly so in thin than in fat people; unless as deepening the voice and either lessening or increasing fulness of person, age appears to have no influence upon its amount.

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* In H. Young, ætat. 40, (carcinoma of ileum and ileo-vesical fistula; U. C. H., Females, vol. vii. pp. 130), approximation of the second and third left cartilages was noticed on full inspiration. The left lung was healthy, weighing only 13·75 oz.; but there were old general adhesions. Also case of Moore, U. C. H., Males, vol. viii. p. 390.

The vibration is scarcely affected by tension or relaxation of the muscles over which the hand is laid: in the great majority of cases it is stronger in recumbency than in the sitting posture (in twenty-two trials, sixteen times greater lying than sitting, four times equal, twice more marked in the sitting than the lying posture). It is greatly more marked when some sounds are uttered than others, and hence the importance, in delicate comparative trials, of making the person examined repeat the same word or words.*

As a general truth, the intensity of the fremitus is considerably greater on the right side of the chest than the left,—the greatest amount of this excess existing in the infra-clavicular, infra-scapular, and inter-scapular regions. Exceptional regions are the right infra-axillary and infra-mammary, where the presence of the liver interrupts the vibrations, and throws into comparative prominence the naturally weak fremitus in the corresponding regions on the left side: the difference would be greater, were it not for the presence of the spleen in the latter position. Where the heart is uncovered by the lung, vibration is totally absent, and the right edge of that space may be traced by its abrupt cessation there; over the left lung there is naturally so little vocal vibration, that modifications of the sign can scarcely be used with confidence for making out the left edge of the space. The lower border of the right lung may be traced by the abrupt cessation of all fremitus immediately below it.

The fremitus is intensely marked over the larynx and trachea, stronger at the sternal than the humeral halves of the infra-clavicular regions, generally faintly manifest on the right clavicle, and imperceptible at the top of the sternum.

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* In singing, the fremitus is much more marked when the voice is bass, baritone, or contr'alto, than when tenor or soprano; and it accompanies the lower notes of any given register to a much greater amount than the upper; it may be absolutely null on a high note, though most loudly sounded, while it is well marked with a low note of the same voice softly uttered. From a few trials I find that the fremitus ceases with soprano and mezzo-soprano voices between *f* and *a* on the lines. Baritones either retain the fremitus through their whole register, or lose it about their upper *f*. The supra-laryngeal register of all voices is without fremitus.

45. The natural vocal fremitus is susceptible, *in disease*, of *increase* or *diminution*. As in the case of other signs, the existing amount of change is most effectually ascertained by comparison of the two sides of the chest; but in making this comparison, the observer must carefully bear in mind the great differences naturally existing on these two sides. Unnatural density of the pulmonary texture, produced by solid infiltration, *unless this be carried to an extreme amount*, intensifies vocal vibration,—as in tuberculous or plastic infiltration, acute or chronic: pneumonia of the left base posteriorly will thus raise the fremitus above the standard of the right base in health.* Pulmonary apoplexy and œdema act, within my experience, in the same way, but to a slight amount. In dilatation of the bronchi the increased calibre of the vibrating tubes, as well as adjacent consolidation, commonly tend to the same result. In pleuritic effusion occupying the lower part of the side, the infra-clavicular region may furnish fremitus in excess.

When the lung-substance is removed to a distance from the chest-wall by gaseous or liquid accumulation in the pleura, as in pneumo-thorax and in pleural effusions, dropsical, hæmorrhagic or inflammatory, the fremitus is, as a rule, annulled. Unfortunately, however, the fremitus is, in some cases, retained at the right base, even when a considerable amount of fluid exists in the pleura. Sometimes explicable by solid adhesions conveying the vibrations from the lung to the chest wall, in other instances the anomaly does not admit of explanation. The theories of unison-resonance, echo, and consonance, do not fairly meet the difficulty. The influence of solid accumulation, either in the lung or pleura, varies according to circumstances: *very extensive* lung-infiltration, whether fibrino-plastic, pseudo-fibrous, carcinomatous or other, deadens the vibration, especially if the infiltrated parts be distant from the larger bronchi, and the voice feeble; extra-pulmonary tumors and aneurisms produce the same effect. But if the other circumstances, such as the strength and graveness of the voice, be favourable, aneurisms, tumors and cancerous infiltrations will not, even when of tolerable size, annihilate fremitus, if they be in close connection with the larger bronchi. It is commonly said that in vesicular emphysema the vibration is impaired; I have not found this habitual, and in some cases its intensity is above the range of health.

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* In cases of universal and very dense acute tuberculous infiltration the fremitus may be strong superiorly near the large tubes, less distinct inferiorly. (Case of Hodson, U. C. H., Males, vol. ix. p. 17.)

45.* Vocal fremitus and audible vocal resonance bear no uniform relationship to each other either in health or disease: for though, when the voice is grave, and low notes are sounded, they both increase and decrease equably, under the converse circumstances audible resonance may be powerful, while the fremitus is feeble or null.

As a general rule, sounds of high pitch give *relatively* most audible resonance, sounds of low pitch most fremitus. But the exceptions to this rule in the acoustics of the chest are numerous. An individual with gruff low-pitched voice may have powerful bronchophony in a spot where the fremitus is next to null.*

46. The act of coughing produces a vibration similar to, but less marked than the vocal. This vibration (*tussive fremitus*) suffers the same kinds of modification in disease; but is valueless clinically, unless in cases of aphonia.

47. Certain rhonchi throw the bronchial tubes into vibration sufficiently strong to be felt on the surface of the chest (*rhonchal fremitus*): the sibilant, sonorous, and mucous, have all this property. The cavernous rhonchus, produced in excavations of the lung near the surface, may be accompanied with marked fremitus, and without fluctuation being perceptible to the finger. Stridulous respiration even, such as that attending

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* Crowhurst (right pleuritic effusion), U. C. H., Males, vol. ix. p. 142.

aneurismal pressure on the trachea, may produce very distinct fremitus, greater in inspiration than in expiration.

48. In the natural state of the pleura, the gliding motion of its costal and pulmonary laminæ upon each other gives rise to no vibration perceptible by the hand applied to the surface. Nor is it usual, even in cases where audible friction-phenomena exist, to discover such vibration: in some instances, however, it may be detected; and the sensation conveyed (*friction-fremitus*), though distinctly somewhat vibratile, nevertheless possesses more of a simply rubbing character, just as might be anticipated from a consideration of its cause,—the collision of plastic matter on the pleural surfaces. In point of intensity it varies greatly; in some cases perceptible, even in ordinary breathing, to the patient himself,—in others it is only evolved by forced inspiration, and only to be caught occasionally. Accompanying either the inspiration or expiration-movement, it is more commonly associated with the former. I have met with it to a higher degree at the absorption-period, than at the outset, of pleurisy.

49. A pulsatile movement of the lung, attended with a quasi-tremor on the surface of the chest, and synchronous with the systole of the heart, has been noticed, first by Dr. Graves, in some rare instances of pneumonia and intra-thoracic cancer. Here, too, may be included the impulse of pulsating empyema.

50. Palpation may also be used for the detection of fluids contained either in the lungs or pleura. The sensation is that of ordinary fluctuation, commonly attended with a certain degree of vibratile tremor. Its existence may be ascertained, either by the movements of the fingers used by surgeons for detecting fluid in an abscess (*simple fluctuation*); or succussion of the chest may be required (*fluctuation by succussion*); or percussion of the surface, be necessary to produce it ("*peripheric*" *fluctuation*); or it may occur through the influence of respiration as an attendant on cavernous rhonchi (*rhonchal fluctuation*): in this latter case vibration may be more distinctly felt than in the

others. The "peripheric" species, described by Dr. C. Tarral, is to be detected by giving a quick sharp fillip in an intercostal space, perpendicular to the surface, when a sensation of fluctuation will be transmitted to a finger of the other hand firmly applied to the surface in the same space, at a short distance from the point percussed. Simple fluctuation is producible, where the intercostal spaces are much bulged out by pleuritic effusion; peripheric fluctuation in the same cases, but most perfectly where air and fluid co-exist in the pleura; fluctuation by succussion in cases of hydro-pneumo-thorax and of large excavation in the lung.

51. Application of the hand to the surface, below the clavicles in the female, below the epigastrium in the male, is the readiest way of ascertaining the frequency of respiration.

52. The absolute frequency of respiration in health averages in the waking adult twenty per minute; greatest at birth, it decreases quinquennially to the age of thirty, and between this period and the fiftieth year again increases somewhat.* Respiration is slightly quickened by fulness of the stomach, and is slower in the male than the female, in the lying than in the sitting, and in the sitting than in the standing postures.

In disease the absolute frequency may rise to eighty, and even upwards, and setting aside moribund persons, may fall to eight.

53. The ratio of the respiration to the pulse is more important, in diagnosis, than the absolute frequency of either. In the adult the natural ratio lies between 1 : 4 and 1 : 4·5. The slower the pulse absolutely, the higher the ratio of the respiration; a pulse of sixty will relatively give a larger number of respirations, than one of eighty. The influence of posture is relatively greater on respiration than on the pulse; hence the ratio varies in the same individual in the three chief postures.

In disease the pulse-respiration ratio is subject to remarkable perversions, which are sometimes of signal use in diagnosis. The extreme ratios which have fallen under my own notice are as 9:1 in chorea,* and as 1·7 :1 in pneumonia. These perversions may be maintained, whether the pulse be, absolutely considered, frequent or not. In hysteria the ratio may range 5:1 to 1· 8:1.

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* Quetelet. Sur l'Homme, t. ii. p. 91. 1836.

SECTION III.—MENSURATION.

54. The object of measuring the chest is twofold: First, to ascertain, more accurately than can be done by inspection and application of the hand, the comparative bulk of the two sides, the relative positions of their different parts, and, in some few instances, the distances between those parts and certain fixed points beyond the limits of the thorax (*measurements in rest*): Secondly, to estimate with precision the amount of expansion and retraction of the chest accompanying inspiration and expiration (*measurements in motion*).

§ I.—Mensuration in Rest.

55. A complete system of Mensuration would comprise the following admeasurements:—

A. General.—(*a*) Circular. 1. *On the level of the sixth cartilage;* 2. Midway between the nipples and clavicles. (*b*) Transverse; 3. From the point of one acromion to that of the other; 4. In the axillæ; 5. At the base of the chest. (*c*) Antero-posterior. 6. *Under the clavicles;* 7. At the base of the chest. (*d*) Vertical. 8. *From the clavicle to the most dependant point of ribs.*

B. Partial.—(*a*) Horizontal. 1. *From the nipple to the middle line of the sternum.* (*b*) Vertical. 2. From the middle of the sternal notch to the nipple; 3. From the nipple to the antero-superior spine of the ileum; 4. From the most dependent point of the twelfth rib to the same spine.

56. But these different kinds of measurement are not all of equal importance, especially in the present state of knowledge,

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* Case of Carpenter, *Clin. Lect.* "Lancet," January, 1849.

—either because some of them really convey information of very secondary value, or because they have not as yet been sufficiently practised to render the physician familiar with the indications derivable from them. The measurements which it is of real consequence for the student to understand, and in all doubtful cases to practise, are distinguished by italics: at the same time, it would be a mistake to imagine that all the others may not occasionally furnish more or less useful information, either confirmatory or corrective of results otherwise obtained.

57. A. GENERAL MEASUREMENTS.—(*a*) *Circular*, (1.) *on the level of the sixth costo-sternal joint.*—Circular measurement of the chest, as commonly performed with a *single* graduated tape passed round the thorax from the middle line anteriorly, is a troublesome process, requiring the patient to be raised to the sitting posture, and the co-operation of two persons. Besides, the difficulty of ascertaining the precise point of the measure, corresponding to the spine, renders the process inaccurate. These difficulties have been removed by a very simple plan, suggested, I believe, by Dr. Hare,—that of joining together *two* such tapes at the commencement of their scales, and fixing them, as the patient reclines, at their line of union, to the spine: each side of the chest has thus its separate measure. By padding the inner surface of both measures, close to their line of junction, a sort of saddle is made, which readily fixes itself to the spinous processes.

58. The circular width of the chest varies so widely in healthy individuals, that there is little practical utility in attempting to fix its mean value. I have known it in the adult male so high, on the level of the sixth cartilage, as forty-four inches, and so low as twenty-seven. Perhaps thirty-three inches may be mentioned as the fairest adult average; but the width varies with age, long after the height has become stationary, gradually increasing from the age of sixteen to sixty: so that the mean being thirty inches from the age of sixteen to twenty, it is thirty-four from that of fifty-one to sixty. The circumference

increases, but not in any fixed proportion, with the robustness, stoutness, and height of individuals. The female circumference is, absolutely and relatively, less than the male.

According to M. Woillez, the circumference is greatest in persons following trades that require active exertion of the whole frame, but not of the upper extremities in particular. Far from this, were his number of cases sufficient for the final decision of the question, the latter class of artisans must be held to have the lowest average circumference of thorax. But the absolute measurement is of less importance than might appear, for width is not an index of expansile power; on the contrary, these statical and dynamical conditions may, in fat people especially, be inversely as each other.

59. The relationship of pectoral to abdominal circumference varies with age: in infancy and childhood the latter is greater than the former. In the male adult, less so in the female, the chest exceeds the abdomen in width. In cases of abdominal obesity the natural ratio becomes perverted.

60. Certain definite relationships exist between the girth of the chest on the one hand, and the width of the shoulders, the distance between the nipples and the antero-posterior diameter on the other (Brent); but as their perversions do not furnish any precise diagnostic inferences, it is unnecessary to describe them.

61. The two sides of the chest are of unequal semi-circumference in about five-sixths of healthy adults; a mean excess of about half an inch existing on the right side in right-handed individuals;* in left-handed persons the left side sometimes measures more than, or more frequently the same as, the right. These propositions hold true of both sexes; but the difference is slightly greater in males than females. In infancy and youth the two sides scarcely differ. I have not traced any special influence of trade on these measurements; but accidental circumstances, unconnected with disease of the thoracic organs,

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* This excess becomes the more remarkable, when the frequency of slight convexity of the dorsal spine to the right, in health, is considered.

are liable to modify their ratio. Thus, distension of the stomach with flatus or food may equalise the measurement, for the time it lasts, on the two sides; and I am inclined to believe, that the deficiency of motion, kept up by fractured ribs in some cases for a very lengthened period, perhaps for a life-time, may in the end diminish the dimensions of the side,—the lung probably, just as in cases where its expansion and retraction are interfered with by pressure of a tumor on a main bronchus, collapsing and becoming more or less atrophous, while the thoracic walls fall in proportionally.*

62. The most important *morbid conditions* of circular dimensions are *increase* or *diminution of width* of either side, as compared with the other. They occur respectively in the same diseases, already enumerated under these heads, as morbid expansion and retraction.

63. *Of circular width midway between the nipple and clavicle* little has been ascertained. The scapulæ prevent the real measures from being taken, and in some persons raise the circumference here above that on the level of the sixth cartilage; as far as is now known, the ratio of the upper and lower circumferences does not appear to me sufficiently constant to be trusted to clinically. It is matter of general belief that the size of the upper part of the chest, compared with that of the base, is greater in proportion as the muscular and osseous systems, especially the latter, are strongly developed, and the constitution of the individual free from the taint of predisposition to phthisis. Though not prepared to say positively whether the excess of width, existing at the lower part of the chest on the right side, holds in the upper regions also, I am inclined, from a limited number of observations, to believe that it does, though to a less amount than inferiorly.

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* Case of Bassett (U. C. H., Males, vol. ii. p. 214); the right side (the man being right-handed and free from pulmonary disease, capable of explaining the fact), measured opposite the sixth costo-sternal joint $16\frac{7}{8}$ inches, the left $18\frac{3}{8}$ inches: he had old fractured ribs on the right side.

64. (*b*) *Transverse.*—Respecting mensuration of the transverse diameters of the chest, I have no precise information to offer. It should be made with a pair of callipers, and there can be little question that diagnostic data of importance might occasionally be derived from its employment. It would, however, in the greater number of cases, do little more than confirm the results of inspection; for diminution of the transverse diameter, in respect of the antero-posterior, the change which, it may be presumed, would most frequently present itself, is one of those alterations of shape which most readily attract the eye.

65. (*c*) *Antero-posterior.*—A pair of common steel callipers is the simplest instrument for determining the antero-posterior diameter of the chest. The absolute maximum measurement varies widely,—the extremes I have actually noted in males of middle height, free from chest-disease, being eight and twelve inches. The difficulties, in comparing the diameters of the two sides, are to apply the blades of the callipers with exactly the same force, and (a far from easy task, on account of the slope of the surface of the chest), to exactly corresponding points on both sides.

In measuring the antero-posterior diameter of the apex of the chest on either side, the extremity of one blade should be placed immediately under the centre of the clavicle, the other upon the corresponding point of the spine of the scapula, the equi-distance of both extremities from the middle line being at the same time carefully ensured. The diameter of the right side in this situation, as also over the sixth rib, will be found, in the greater number of healthy persons, to exceed that of the left (possibly as a compensation for the relatively less height of the right lung), but by so very small an amount that, where an excess at all marked is detected on that side, the physiological disparity may be ignored. In other words, an excess of even a fourth of an inch on the right side, furnishes sufficient evidence of morbid depression or diminished diameter on the left; though the existence of a similar excess on the left side will be still more strongly conclusive of contraction on the right.

66. The *morbid states* discoverable by the measurement now described are, *diminution* and *increase* of the antero-posterior diameter. The latter change occurs in pleurisy with effusion, pneumonia, hypertrophy of the lung, emphysema, intra-thoracic tumor and aneurism, various cardiac affections, acute general tuberculisation of a lung, and probably chronic tuberculous disease of the apex, at its very earliest period. Diminution, on the other hand, arises in the more advanced stages of tubercle, in simple chronic consolidation, in the absorption-period of pleurisy with retraction, and in cases of prolonged mechanical obstruction to the entry of air through the larynx or main bronchi.

67. (*d*) *Vertical.*—The vertical measurement of the chest has hitherto been only practised in front; measured with a tape, the distance between the centre of the clavicle and the most dependent point of the corresponding ribs is found to be very closely the same on both sides.

68. This distance is liable to be *increased* in cases of solid, liquid, or gaseous accumulation in the chest; to be *diminished* in chronic pleurisy with retraction. But though elevation of the diaphragm, with consequent diminished vertical height of the thoracic *cavity* on either side, and also the converse states are not uncommon, changes of the measures on the *surface* are rare.

69. B. PARTIAL MEASUREMENTS.—(*a*) *Horizontal. From the nipple to the middle line.* The nipples are equi-distant from the middle line in the healthy adult male. The distance between either of them and that line is liable in disease to increase, and more frequently to diminution. Hypertrophy of the heart, pericardial effusion, mediastinal tumors and aneurisms in certain situations increase it, though not often seriously; on the other hand, diminution in cases of retraction after pleurisy, varying in amount from a quarter of an inch to an inch and a quarter, may be detected.

70. (*b*) *Vertical. From the middle of the notch of the sternum to the nipple.*—These points are equi-distant on the two sides in chests of perfectly regular form. We have already seen, however, that normally the left nipple sometimes lies lower than the right; hence the space comprised between the nipple and clavicle on that side may be greater than on the other, independently of the influence of disease.

71. The only *morbid variation* observed in this measurement is *increase*, and it is obvious, from what has just been said, that this sign will have more value on the right than the left side. And mensuration is less useful in respect of this sign than inspection; for, on account of the flattening of the surface, which commonly coexists with lowered position of the nipple from disease, the superficial measurement undergoes a diminution which may more than compensate for the increase produced by the latter cause. In a remarkable example of this apparent contradiction between the results of inspection and mensuration, although the left nipple was manifestly lower than the right, the distance between the former and its corresponding sterno-clavicular joint was only 5½ inches, while that between the same joints on the right side was 6¼ inches.* It is to be supposed that such will usually be the result of mensuration, where the retraction after pleurisy affects more especially the antero-posterior diameter.

72. The distances comprised between the *nipple and the antero-superior spine of the ileum*, and between the *most dependent point of the twelfth rib and the same process*, are equal on the two sides in health. They undergo *diminution* on either side in cases of marked retraction of the chest; and, probably, *increase* in those of expansion.

§ II.—Mensuration in Movement.

73. There are two plans for submitting to measurement the influence of respiration on the dimensions of the chest; the one

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* Cyclopædia of Surgery, article *Empyema*, p. 102.

estimates the amount of antero-posterior movement, the other the amount of expansion and retraction, attending the act.

74. The former measurement is made by an instrument of very ingenious construction, named by its inventor, Dr. Sibson,* the "Chest-Measurer." In principle a callipers, of which one branch is movable, this instrument is capable, by means of an index and dial fixed to its movable branch, of indicating any change in the antero-posterior diameter of the chest or abdomen, even to the one-hundredth part of an inch, and has enabled Dr. Sibson to establish the following, among numerous other, propositions concerning the respiratory movements in health. In the robust male the forward movement of the sternum and of the seven upper ribs ranges from one-fiftieth to one-fourteenth of an inch in ordinary inspiration, and from half an inch to two inches during deep inspiration.† On the five lower ribs the ordinary movement is less, and the forced movement greater, than over the upper seven. The movement is somewhat less on the left side than the right, below the second rib. The ordinary abdominal movement is about a quarter of an inch; the extreme ranging from about half an inch to an inch and a half.

Unless the expansibility of the chest be directly as the forward motion of the anterior parts of the ribs, the indications of the "Chest-Measurer" do not accurately express variations in the amount of the former. This is seen in health. The instrument shows, according to its inventor, that the forward motion is in the male greatest, during calm breathing, superiorly, least inferiorly; while, during forced breathing, the lower ribs play relatively more than the upper: now the eye and circular measurement teach us that the exact reverse is the truth in regard of the really important clinical fact—expansile action [30.] Again, when the costal cartilages are stiffened by age or precocious ossification, the expansion may be greater materially than in the ratio of forward costal motion ; the converse state of things will exist in youth. In disease, too, the forward motion of the chest, and its lateral expansion, may be very differently affected : in a case of large excavation under the left, and consolidation with small excavations under the right, clavicle, the lower part of the sternum and adjoining cartilages *receded* visibly during inspiration, yet a fair amount of *circular expansion* was produced by the act.* The deceptive influence of the torsion-movement of the ribs (which will apparently increase or decrease the amount of their forward motion, according as the movable rack is fixed near their lower or upper edge) must be borne in mind ; the very delicacy of the instrument might otherwise mar its utility. But in localising with precision deficiency or excess of antero-posterior motion, and in estimating changes from day to day in the amount of either, the indications of the "Chest-Measurer" are greatly superior in perfection, it need scarcely be added, to those furnished by application of the hand.

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* Med. Chir. Trans. vol. xxxi., p. 353. A modification of the instrument has been proposed by Dr. Quain.

† Haller, estimating the effects of a moderate inspiration, found the forward movement of the sternum, superiorly, 2½ lines, inferiorly from 3 to 8 lines.

75. The amount of inspiratory expansion and expiratory retraction of the chest and abdomen is measurable by the double tape already mentioned.† Applied closely, but not tightly, to the chest, on the level of the sixth cartilage, the tape shows that the expansion accompanying ordinary calm inspiration in health is very slight, averaging about a quarter of an inch in the healthy male adult, with a circumference of thirty-three inches : this amount is, practically speaking, shared equally by the two sides. In forced inspiration the circumference is increased from the medium or tranquil state by from one-and-a-half to three inches,

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* Case of Green (U. C. H., Females, vol. v. p. 146, July, 1850). The same phenomenon sometimes occurs in pleuritic effusion also.

† Mr. Henry Thompson, of University College, has recently suggested a very simple addition to the tape-measure, whereby the absolute and relative expansion of the two sides of the chest may be ascertained during *one* and the same respiration. The more ordinary plan requires two ; and as no two respirations are probably precisely equal, Mr. Thompson's instrument, which may be had of Coxeter, Grafton Street, obviates a source of fallacy.

and is somewhat greater on the right than on the left side,—the deficiency on the latter mainly depending on the heart. The total circular difference between forced inspiration and forced expiration ranges between two-and-a-half and five inches: this is estimated by taking admeasurements at the moments the patient has been made first to fill, and then to empty, the chest to the fullest possible amount. At the same time the difference in the respective extremes on the two sides is seen. But the same amount of difference may exist between extreme inspiration and extreme expiration on the two sides, and yet be very differently produced: it may, on one side, depend in the main on great expansion above, and on the other on great retraction below, the *medium state;* in other words, inspiration may be free to excess on the former, expiration on the latter, side. In health, however, such want of harmony in the expiratory and inspiratory efficiency on the two sides is never met with, except to the very slightest calculable amount, and is probably traceable to the difficulty of the observation.

The chest-play is remarkably under the influence of posture; least in decumbency, it increases successively, chiefly in its inspiratory division, in the sitting and standing postures,—very much in the ratio of the increasing frequency of respiration.

76. Forced breathing has scarcely any influence in expanding the abdomen: in a healthy male adult, now under observation, five feet six inches in height, in whom, while standing, extreme expiration gives a circumference opposite the sixth cartilage of 29½ inches, and extreme inspiration, one of 34 inches (a very rare amount of thoracic play, especially with so low a stature), the abdominal inspiratory expansion equals only a quarter of an inch.

77. *In disease*, mensuration ascertains with accuracy the amount of deficiency of expansion on both sides, and on one side as compared with the other, the mere existence of which deficiency is more or less readily ascertained by the sight and touch. The Section on Inspection [31] may therefore be

referred to for a list of the affections in which deficient expansion is to be estimated by measure.

The variations from the healthy standard thus discoverable are sufficiently striking. In chronic empyema, for instance, the total difference between the fullest expiration and the fullest inspiration on the affected side, may scarcely reach one-sixteenth of an inch; while the other side, especially if time has elapsed for its lung to grow hypertrophous, may have a play, as observed, of nearly two and a half inches,—an amount reaching the limits of health for both sides united. Hemiplegia will materially lower the respiratory play on the affected side: thus, in two cases elsewhere described,* the range of motion on the paralysed side equalled in each about a quarter of an inch, while that on the non-affected side measured in one instance three-quarters of an inch, and in the other an inch.

78. When the respiration-play of both sides combined does not reach two and a half inches, disease impeding respiration in all probability exists: and when the total amount being equal to, or exceeding, a healthy average, the shares of the two sides are notably unequal, disease exists, impairing the play of one side, and exaggerating that of the other.

79. But the measured range between forced expiration and forced inspiration may be the same on the two sides, and yet the accompanying change of volume of the two lungs be very different in kind. On the one side the play may be chiefly effected by the ready inspiratory expansion of the lung,—on the other, by the great efficiency of expiratory contraction. In the former case, the lung maintains its relative efficiency by its power of taking in beyond its medium quantity of air: in the latter by its power of expressing air, which in ordinary breathing stagnates within it; in the former case expansibility, in the latter elasticity, predominates. Here are two very different conditions of lungs, most important to be distinguished, and which have hitherto never been made the subject of clinical

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* *Clinical Lectures*, "Lancet," March 17, 1849.

study. The difficulty of the study is, it is true, extreme, from the nicety required in fixing the standard of comparison, namely, the medium or tranquil measurement of the chest. My observations on this matter are as yet too limited to justify me in announcing general conclusions; but they prove to me that with care the inquiry may become rich in results. It seems almost a matter of necessity that, as the difference of power in the two cases directly bears on different portions of respiratory air, the influence on the oxygenating process must vary in each. Where the expiratory force is in excess, the "supplementary" air [82], where the inspiratory action predominates, the "complementary" air, must be severally most affected.

80. The forced breathing of health expands the chest in both sexes and at all ages, out of all proportion with the abdomen. In the forced breathing of diseases seriously affecting thoracic expansion, the diaphragm on the contrary assumes unusual energy, descends more than natural and expands the abdomen in excess. Severe pleurodynia will suffice to transpose the respiration-movements in this way; I have known the pain attending herpes zoster do so to a slight extent. Where local rheumatism affects the parietes of both the abdomen and chest, it is curious to observe the medium state of modification in the relationship of abdominal and thoracic expansion in forced breathing: the pectoral expansion is relatively somewhat less, the abdominal somewhat more, than the healthy average.*

§ III.—Mensuration of Capacity for Air.

81. Although the results of mensuration in movement may be taken as fair indices of the relative amounts of air circulating in the lungs of different individuals, and of the same individual at different times, it is plain they give no idea of absolute amounts. But direct attempts have been made with the help of various instruments, to fix the entire volume of air admitted

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* Case of William Farrett; U. C. H., Males, vol. vi. Nov. 1850.

into the lungs, under varying conditions of health and of disease.

82. Now the air habitually present in, or capable of being introduced by respiration into, the lungs, is divisible into various quantities. First, there is a volume of air which remains in the cells even after the complete collapse of the lungs in the dead body, and which cannot, by ordinary means at least, be artificially expressed from their tissue, though thoroughly removable by the compressing influence of certain diseases: this may be called *persistent* air. Secondly, a volume, which, though collapse expels it after death, cannot be expressed from the lungs during life by the strongest expiratory effort; this may be termed *residual* air.* Thirdly, the volume of air, which can be expelled by a forced, after an ordinary, expiration, may be termed *supplementary*. Fourthly, the *breath*, or *tidal* air, that which in flux and reflux passes to and fro in calm breathing. Fifthly, the volume which can be inhaled by a forced, after an ordinary, inspiration,—and may be given the name *complementary*. The persistent, residual, and supplementary airs together, may be styled *stagnant* air; while the sum of the supplementary, tidal, and complementary quantities, may be named *total breathing volume*. Tabularly, they would stand thus:—

Stagnant = Persistent
Residual
Supplementary
Tidal = Total breathing volume.
Complementary

83. The earlier attempts at estimating these volumes, either singly or in combination, varied so ludicrously, that to obtain

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* I here adopt, for the most part, the terms devised by Mr. Jeffreys ("Statics of the Chest," Lond. 1843.) The distinction made in the text between persistent and residual air, has not, so far as I know, been hitherto recognised; but it is evidently real. Of course it is the volume only, not the actual same air, that is persistent: the fact of such persistence, shows the vital importance of the law of diffusion of gases, as unravelled by Mr. Graham; were it not for that law, scarcely any fresh air, at least with the existing mechanics of respiration, could reach the air-cells for the purposes of blood-ventilation.

physiological standards seemed hopeless. Healthy types proving unattainable, the application of pulmometry to the clinical study of disease, could not, in spite of the efforts of Kentish and Herbst, prove successful. And yet these observers, more acute than their predecessors, recognised the influence of various collateral conditions on the breathing volumes. Others have since done so even more fully; still the most perfect results are, it is to be feared, only approximatively true.

84. Thus M. Bourgery,* investigating what he terms the "Measure of respiration," which proves to be the compound of the tidal and complementary airs, finds this volume deeply influenced by age, sex, and leanness, or the reverse, of person: health and vigour of body do not compensate for youth and thinness. The function reaches its maximum at the age of thirty; † at this period a forced inspiration will add to the stagnant air 2·5 to 4·3 litres in the male, from 1·1 to 2·2 litres in the female.‡ The boy of fifteen can inspire 2 litres, the man of eighty but 1·35.

85. Dr. Hutchinson,§ in turn, studying by means of his valuable instrument, the Spirometer, the quantity of air which may be expelled from the chest by the fullest possible expiration, succeeding the fullest possible inspiration (that is the sum of the supplementary, tidal, and complementary airs, or the total breathing-volume), affirms that the physical conditions dominating all others, which influence breathing, are stature and weight. His experiments lead to the inference, that the total breathing-

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* Mém. de l'Acad. des Sciences, Janv. 1843.

† This discovery gives incidental proof that there is not any direct ratio between the breathing-volume and the girth of the chest; for, as before stated, [58] the circular width increases with advancing years.

‡ The influence of sex was, however, originally ascertained by Thrackrah, who showed that "while healthy men inhale by the pulmometer 200 cubic inches and upwards, women rarely exceeded 100, and often do not reach that amount." (Effects of Arts, &c., 2nd edit. p. 181. 1832.)

§ Med. Chir. Trans. vol. xxix. 1846. A simple spirometer, accurate in its measurements, and easily carried in the pocket, has recently been invented by Coxeter, of Grafton Street, and is sold at a small price.

volume varies in a definite and calculable ratio with the height of the individual: that the mean for the male, at the height of five feet, being 174 cubic inches, it increases, at 60° Fah., with every inch of stature between five and six feet, by eight additional cubic inches. Evidence seems to have been collected on this point sufficient to show the generality of the rule, but not the universality of the law.*

86. Are these physiological results applicable as aids to thoracic diagnosis? Rudely,—not with precision. For, M. Bourgery's mean general standards for sex, age, and varying leanness of person, and Dr. Hutchinson's for stature, being admitted, the question arises, what oscillations above and below those standards are compatible with health? Now not only can no positive reply at the present day be given, but such is the complexity of the problem, it seems unlikely ever to be furnished.

The calculation has, no doubt, been offered for Dr. Hutchinson's results, that a deficiency of 16 per 100 is suspicious, but may possibly arise from physiological peculiarity,—that beyond this, the deficiency is morbid. But in clinical practice it turns out, that the *general* standard of height, even with this correction, is often valueless,—that the *individual* healthy standard often varies far too widely on either side of the general one. So much so, that a great fall may have taken place, from disease, in the breathing-volume of an individual, at a time when he expels a quantity of air above the average standard of men of his height: according to the general standard he is more than healthy, he is extra-capacious; according to his own, he is diseased. For certainty of observation, the individual standard is required; the present man must be compared with the past man, and not with other men. It may be remembered too, that fall below the general average, is a surer indication of disease, than the maintenance of that average, or even of a slight excess, is of health.

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* Kentish, the inventor of the pulmometer, was well aware of the influence of height on breathing-volume ("Account of Baths, &c., with a Description of a Pulmometer," pp. 87, 91, 116, London, 1814); more recently Herbst categorically stated its importance (Ueber die capacität der Lungen für Luft im gesunden und kranken Zustande, Meckel's Archiv. 1828, pp. 98, 101). But neither of these observers attempted to deduce a precise ratio between capacity and height.

But of what disease? Obviously of any disease, whether situated in the lungs and appendages, the heart or great vessels, the abdomen, the encephalon or cord, which interferes on vital or mechanical principles with the expansion or retraction of the lungs. The spirometer indicates when the lungs receive an insufficient supply of air, but gives no inkling of the cause of the deficiency,—unless observation should prove, and of this there seems no present probability, that special scales of reduction of breathing-volume obtain in particular diseases. The spirometer, too, tells nothing of the distribution of the air inspired,—in this view clinically inferior even to semicircular and antero-posterior mensuration, which point out the side and the region receiving too much or too little air.

87. For these and other reasons the spirometer affords no help, where the presence of pulmonary disease being certain, its nature remains an insoluble problem by other methods of physical diagnosis. The weight of the allegation that a fall of breathing-volume, eventually traced to tuberculisation, has been detected at a time, when no ordinary physical sign of pulmonary disease existed, will be elsewhere considered.

88. On the other hand, where the object is to ascertain roughly and rapidly the soundness of a body of men, as in the instance of recruits, the indications of the spirometer may, with practice on the part of the observer, be accepted as significant.*

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* The practical sources of fallacy in the use of this instrument are numerous. Some persons cannot be taught to inspire properly; others to use all the air they actually inhale: some from simple indolence and indifference fail to "blow" an amount they are well capable of; others stimulated to extra-laborious effort, in the hope of proving themselves better than they really feel, mark higher in the scale than many with more capacious lungs: some by habit learn, after a time, to utilise the total volume, a great part of which

In this point of view would the variations of the tidal air, furnish data of greater importance than those of the total breathing-volume? Possibly; no proof exists at least that because an individual can under an effort take in a surprising quantity of air, he therefore habitually circulates and consumes the volume necessary for the maintenance of health. Mr. Jeffreys has calculated that in the healthy adult male this quantity oscillates between 16 and 40, and averages 26, cubic inches; and Vierordt found the amount in his own person range between 10·8 and 42·6 cubic inches.* The wide range compatible with health points to the infinite difficulty, it is to be feared the impossibility, of obtaining serviceable results in disease.

89. The muscular force of expiration is considerably greater in the forced breathing of health than that of inspiration, in about the ratio of 8·03 : 5·68 according to Valentin. Of their relative power in calm breathing we know nothing.

The inspiratory power sometimes exceeds the expiratory in disease [79.]

SECTION IV.—PERCUSSION.

PERCUSSION IN HEALTH.

90. The act of striking the external surface of the chest for purposes of diagnosis is called *percussion;* and the immediate object of the process is the determination of the density of subjacent parts. Applied to the thorax, it serves to establish, by inference, any increase or diminution of the quantity of air naturally contained within that cavity. Statically it tests the quantity of stagnant air,—dynamically, the amount of tidal and complementary air, the lungs contain or receive [82]. It

in early trials was lost (just as persons endowed with even the humblest vocal faculty acquire by practice greatly increased power of sustaining notes); while others, with equal habit are, from mere stupidity, as bad "blowers" at last as at first.

* Vide his article *Respiration,* in Wagner's Handwörterbuch, p. 835. 1844.

indicates also the presence of air in unnatural localities within the thorax.

91. The amount of density is inferred from, (*a*) The nature of the *sound* elicited by percussion; (*b*) The *degree of resistance*, in other words, the elasticity, of the body percussed.

92. (*a*) *Sound.*—The properties of percussion-sound, which, varying with the density, and some other physical conditions, of the textures and materials furnishing it, possess practical importance are:—*Its degree of clearness; its duration;* and *its quality.*

93. *Clearness.*—The conditions of percussion-sound commonly described as *clearness*, and its converse, *dulness*, are scarcely capable of being described: they are readily illustrated by percussing, successively, the chest, at its antero-superior part, and the thigh; and the sounds elicited in these two situations, the former *clear*, the latter *dull*, may be used as measures of comparison for the greater number of sounds producible in various parts of the thorax. Although obviously incorrect, the terms *dull* and *clear* are retained in this work; because, in the first place, their practical signification is generally understood; and, in the second, it is extremely difficult, if not actually impossible, to substitute correct scientific expressions for them. They are incorrect; because *dulness* and *clearness* are not terms opposed to each other, either in the common signification of the words, or in an acoustic sense; nor are dulness and clearness * admitted among the properties of sound by natural philosophers; and hence there is this curious contradiction in the works of various writers on physical diagnosis, who preface their volumes with inquiries into the theory of sound, that no such properties as dulness or clearness are ascribed to it, and yet dull and clear

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* The word clear applied to sounds, strictly speaking, means pure. The notes of an instrument are said to be clear, when they are heard singly and purely, uninterfered with by any extraneous vibrations,—the tones of the human voice, when free from huskiness or any other superadded character impairing their singleness. Now, in this sense, no sound elicited from the chest by percussion can be called clear.

sounds are perpetually spoken of in subsequent descriptions. Again, dull sound is used as synonymous with "little" sound, or "no" sound. This is sometimes, but not always, correct; for there is, in point of fact, as intense noise in many so-called dull, as clear, sounds. It is not in *intensity* that the difference which impresses the ear consists, but in *duration* and sometimes in *pitch;* so long as they both last, one may be as intense as the other. Hence it appears that what is practically called clearness is equivalent to continuousness of sound; and dulness, non-continuousness. And as continuousness of sound depends on the elastic, vibratile character of the material furnishing it, the inference follows that when dulness exists, the material struck is either inelastic, or all vibration is suddenly stopped by some extrinsic influence. In clear and dull sounds, too, there is this further difference,—the former, as those of the chest, approach in character to tones; the latter, as of the thigh, are, at least as transmitted to the ear through the air, utterly toneless and mere noises. Under certain circumstances it becomes possible, even, to assign rudely the pitch of the percussion-sounds of the chest,—they become notes. This occurs, more especially when, in addition to other conditions, the ribs are peculiarly favourable, from shape or texture, to vibration. When the percussion-sounds are listened to directly through a solid stethoscope applied to the surface struck, the difference of pitch on different parts of the chest becomes more obvious, than when the sounds are heard through the air.

94. All wet animal textures in a state of relaxation, with the exception of bone and cartilage, which possess a special clear resonance, furnish a dull sound, or rather a mere noise, under percussion. The viscera are indeed, practically speaking, soundless in themselves,—the proper substances of the liver, spleen, kidney, heart, and lung, from which the air has been artificially expressed, do not appreciably differ in regard of this property: all of them are deficient in the molecular elasticity required for continuous sonorousness. Hence the resonance of the lungs, of

which we speak clinically, depends not on their proper tissue, but on the air they contain, and on the construction of the case in which they are contained. The quantity of bone and cartilage entering into the composition of that case, its hollowness, and the thinness of its walls, in comparison with the extent of its cavity, all conduce to the freedom of sonorous vibration. So, again, equal portions of heart-substance and of liver-substance, when similarly percussed, will give out sounds short, abrupt, and toneless, in no wise distinguishable from each other; yet the heart and liver, *in situ*, sound differently: the pitch of the heart-sound is perceptibly higher than that of the liver, and the difference depends at once on the hollowness of the former, and the different properties of the cavities containing the two organs.

95. *Duration.*—Difference of the duration of the sounds emitted by bodies of different kinds under percussion may be illustrated—if examples of the familiar fact be required—by the prolonged ringing sound produced by striking a gong, and the short abrupt one similarly yielded by a mass of putty. The disparity in these two instances is considerably greater than any observable in percussing the human body, but less degrees can readily be conceived; that existing between the sounds emitted by the thigh and the cranium exemplifies one of those degrees.

The duration of the percussion-sound varies also very distinctly in different parts of the chest; for instance, at the upper part of the sternum and over the heart. The greater the dulness, the shorter, we have seen, is the duration of the sound; but as changes in the former are much more readily appreciated than in the latter property, this is not one from which much information is derived in practice.

96. *Quality.*—The quality or timbre of the sound emitted by the chest in health is not easily described; the usual statement, that it is a "good clear" sound, manifestly gives no distinct notion of its nature. It conveys the ideas of softness

and of hollowness to a moderate degree; but is in fact *sui generis*, and a few trials upon a healthy chest will make the student more familiar with it, than could the most laboured description. The healthy quality, which, for brevity's sake, may be called *pulmonary*, is sufficiently marked and peculiar, to render the variations to which it is subject in disease easily perceived.

97. (*b*) *Degree of Resistance.*—When percussing a chest immediately over lung perfectly free from all disease, the observer is conscious of a slight yielding motion on the part of the walls, accompanied with a sensation of elasticity. It is impossible to fix the degree of this elasticity, but the reality of its existence may at once be ascertained by percussing comparatively the anterior part of the thorax and the thigh; in the latter situation a sensation of dead unyielding resistance is experienced. The amount of resistance varies inversely as the clearness of the percussion-sound; and directly as the amount of bone in the walls.

98. Considered in respect of the manner of manipulating, percussion is either *immediate* or *mediate*.

99. *Immediate* percussion, the invention of Avenbrugger, is performed by striking the surface of the chest with the points of the four fingers of the right hand, united into a point on a level with each other, the ball of the thumb being placed firmly against the index finger opposite the articulation of the second with the third phalanx, so as to support and give firmness to the fingers. The hand being thus prepared, the points of the fingers are brought perpendicularly down upon the surface with a sharp and quick stroke, which is found to produce a sound varying in properties with the condition of the subjacent parts. Avenbrugger recommended, as an important precaution, that the patient's chest should be covered with a thin dress, or that the observer should wear a glove,—the object being, by either plan, to prevent the sort of clack resulting from the contact of the naked hand and skin. If, in accordance with the advice of

Laennec, the hand be kept naked and the chest covered, it is very necessary, as pointed out by Avenbrugger and others, that the shirt or other covering be drawn tight over the part percussed. Immediate percussion may also be performed by striking the chest with the palmar surface of the fingers: others tap the surface lightly with the small end of the stethoscope; but patients always dislike this, and it may be productive of serious pain.

Immediate percussion has, however, almost completely fallen into disuse, less in consequence of the positive objections to its employment than of the discovery, in mediate percussion, of a plan, if not more ready in its application, much more satisfactory in its results. And, in truth, those objections are, in themselves, far from unimportant. Direct percussion, even when very skilfully performed, rarely fails to give more or less pain: in the hands of an awkward and inexperienced person it is really almost unbearable, and may, with some colour of justice, —because a more efficacious and less painful method can be employed,—be regarded as a piece of cruelty to the patient. Direct percussion cannot be performed over the intercostal spaces; and when the patient is very fat, or the subcutaneous tissues are anasarcous or emphysematous, its results cannot at all be depended upon. Besides, the manual process of immediate percussion is exceedingly difficult of acquirement, and the least inattention to the manner of its performance will scarcely fail of leading to erroneous notions of the state of the chest. Mediate percussion is, no doubt, liable to a certain extent to the same objection, so much as to make its practice much more difficult of attainment than that of auscultation; but there can be no doubt that the chances of error are with it much less numerous and serious. There are, however, a few circumstances under which immediate percussion may still be had recourse to with advantage. Thus, in cases where extensive and notable difference between the two sides exists, rapidly striking them with the palmar surface of the hand will leave no doubt as to the fact; indeed it will disclose the amount, though not the superficial extent, of the alteration of sound, almost as satisfactorily as the more delicate process of mediate percussion. In cases of hepatisation and of pleuritic effusion, where it may be inconvenient to submit the patient to a lengthened examination, this method, therefore, has its utility. Again, it will be found that directly tapping the clavicles and spines of the scapulæ with the points of one or more fingers, or with the bent knuckle of the index-finger, conveys as correct information as mediate percussion of those parts.

100. The distinctive character of *mediate* percussion, for the invention of which we are indebted to M. Piorry, is that some solid body, interposed between the chest and percussing agent, receives the direct impulse of the latter. In mediate percussion, or, as I shall in future call it, simply, percussion, there are two chief things to be considered—the material interposed, and the agent used for striking it.

101. The material interposed, termed a pleximeter (πλῆξις, percussion, and μέτρον, a measure), may be of different kinds. That employed by M. Piorry is a thin, circular, or oval plate of ivory, about an inch and a half in diameter, and provided with two prominences or handles, fixed at right angles to its plane surface, and at nearly opposite points of its circumference; these enable the observer to hold it steadily, and apply it evenly and firmly to the surface. Innumerable have been the modifications of this, and the varieties of new pleximeters, proposed from time to time; of these, a finger of the left hand (Skerrett ?) and a flat piece of india-rubber (Louis), are in my mind decidedly the best. The index or middle finger, on account of their always being within reach, on account of the accuracy with which they may be fitted, as it were, to the various depressions on the surface, and on account of the absence of parade in their employment, will no doubt always continue the pleximeters in most common use. They have in these points of view, an unquestionable superiority over M. Piorry's plate of ivory. The

india-rubber pleximeter may, however, be defended: there is nothing pompous in its appearance, and by a little management it may be accurately applied, even in the intercostal spaces of the thinnest persons. It has, besides, this positive advantage, that it saves the finger of the operator,—no trifling matter, where a very large number of patients are to be examined. And its use implies a saving of pain not only to the operator, but also to the patient, as I ascertained some years ago by a considerable series of comparative trials. Some individuals bear percussion without murmur in this way, who resolutely refuse to allow it, if the finger be used for a pleximeter. The only objection I have ever heard urged against the india-rubber is, that it deadens the sound. This, which would be a valid argument if a single point only of the chest were to be percussed, and a direct inference drawn from the result, has in reality not a particle of force; because inferences are invariably drawn from the comparison of different parts.

Whatever pleximeter be employed, it should be placed in accurate and firm contact with the surface; for this reason it appears advisable to apply the palmar, and not the dorsal, surface of the finger to the chest, when this is the pleximeter used. No extrinsic condition modifies the sound so much as the amount of force with which the pleximeter is applied to the surface; and the finger with its dorsal surface turned to the chest is, in this point of view, comparatively unmanageable. The validity of this objection is, however, not generally felt; M. Louis, among others, very frequently percusses in this way, and Dr. Stokes appears to prefer it. It is certainly, in some cases, easier to apply the dorsal than the palmar surface of the finger uniformly to the part of the chest under examination; but this advantage has always seemed to me much more than counterbalanced by the disadvantage just insisted on.

The finger may be applied parallel to the ribs, or at various angles with them. The former way of placing it is materially the more common; and, as a general rule, is by far the more correct, for by it only can the finger be fitted, in thin persons especially, to the irregularities of the surface. But it is sometimes both convenient and advantageous to vary the direction of the finger; and, as it is next to impossible to place the finger uniformly and equably against the surface in the neighbourhood of the right acromial angle, if it be applied horizontally, fixing it at a variable angle with the ribs becomes a matter of necessity. To obviate the difficulty referred to, some persons stand behind the patient while percussing the upper anterior regions: but when this plan is followed, it becomes as difficult to fix the finger on the left side, as on the right when the physician stands in the usual way in front of the patient; the position is besides open to several other manifest objections.

Useful information may sometimes be obtained by using the four fingers of the left hand, laid firmly and closely on the surface, as a pleximeter. When the anatomical cause of variation of sound is considerable in extent, but slight in degree, there is an obvious advantage in including a space of some size under the pleximeter.

102. Whatever be the pleximeter used, the fingers are commonly employed as the percussing agent. The various hammers and accompanying apparatus, invented in this country and abroad, some of them of an appearance to terrify a timid patient, do not seem to me to possess any kind of superiority to the fingers, and labour under the serious disadvantage of depriving the observer of the indications furnished by the sensation of resistance of the parts percussed. The clinical supremacy of the fingers has never been a moment threatened by any of these elaborate inventions; and it appears consequently unnecessary to describe them.* Generally speaking, the index and median fingers, having their points placed upon exactly the same level,

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* However, some practised observers prefer the hammer; and, on the principle of *audi alteram partem*, I would refer the reader to a valuable paper by Dr. Hughes Bennett, in the "Edinburgh Monthly Journal," Oct. 1850.

and supported, or not, by the thumb with its ball laid upon the outer surface of the former, opposite the articulation of its second and third phalanges, make the best instrument for striking with. But the index-finger alone may be used, especially when gentle percussion only is required, and, generally, therefore in the case of children. Under some circumstances three fingers form a useful modification ; or the knuckle of the index-finger—that is, the joint of the first and second phalanges—may be used with good effect ; in percussing the larynx, the most convenient plan is to fillip with the median finger.

When the four fingers of the left hand are used as the pleximeter, those of the right form the best agent for percussing with. If precision be desirable, the tips of the fingers should be used to strike ; in rough examinations the palmar surface of the right-hand fingers, held in firm extension, may be lightly tapped against the dorsum of the left hand.

103. In the case last referred to, the percussing fingers are made to fall horizontally, the more accurately so the better, upon the surface struck ; under all other circumstances, it is of essential importance that the points of the fingers fall perpendicularly upon the pleximeter. The least variation in this respect is liable to be attended with a difference in the sound elicited.

In the act of percussing, *the movement should spring from the wrist only*, the fore-arm and arm being held perfectly motionless. The pain which beginners cause the patient in many cases, and the uncertainty of the results obtained, in a great degree depend upon the ignorance of the value, or neglect, of this rule : the awkwardness of striking from the elbow, or even the shoulder, as is often done, is a matter of less moment ; though an observant patient will scarcely fail to be impressed unfavourably by it, when he finds himself rather pushed about than percussed. But the essential advantages of this mode of percussing are the nicety with which the force of the blow may be regulated, and hence made precisely equal in any two places it is the object to compare; and the great comparative ease of keeping the percussing fingers at *the same angle* in striking repeatedly the same or different spots. Were this point of manipulation generally attended to, it would be infinitely less common, than it now is, to hear a new and different sound elicited by each of a number of successive blows upon the same place;—a variation, the mere possibility of which constitutes a serious drawback to the utility of percussion, as it is too frequently practised.

The force used in striking should never be great, absolutely considered; but it may be made to vary from the most gentle, to a smart tap, according to the object in view. Generally speaking, gentle percussion is advisable, when we desire to ascertain the amount of density of superficial parts; forcible, when deep-seated tissues are the subject of investigation. Corresponding regions of the chest, which yield sounds of the same clearness and duration when gently struck, may yield sounds materially differing in these respects if forcibly percussed, and *vice versâ;* it is therefore obvious, that both modes should be employed in every instance where accuracy of diagnosis is aimed at.

The blow should be quickly and lightly given, the fingers being withdrawn, or at least all pressure removed, the moment their impulse has been effectually communicated to the surface struck; the vibrations of the surface are thus impeded to the least possible amount. To this precept there is but one exception: in eliciting a particular modification of special character of the sound (*cracked-metal character*), the successful production of which depends materially on the manner of striking, it is advisable to give the impulse slowly and heavily, and allow the fingers to press forcibly on the part for some moments, after it has been given.

104. The posture of a patient undergoing percussion should, unless special circumstances prevent it, be the sitting or the standing. This precept is in accordance with the rules of Laennec, but perhaps his motives for laying it down were not

perfectly well grounded. I cannot say I have found, as he represents to be the case, that "if the patient be in bed, the mattress, still more the pillows, and also thick curtains, always render the sound less;" and even admitting the fact to be so, as the object is to obtain comparative and not absolute results, it would constitute an objection of but slight importance. The difficulty of placing the patient perfectly level in bed (and if he be not so placed, the sound on either side is extremely liable to be modified), together with the constrained positions the physician is obliged to place himself in, in order to get at different parts of the chest, appear to me to constitute more solid objections to the recumbent posture.

While the anterior regions are under examination, the patient must hold his head erect, and allow his arms to hang loosely by his sides; his hands may be clasped across the head, to facilitate percussion of the lateral regions; and he should cross his arms pretty tightly in front, and bend his head slightly forwards while the back is examined.

105. Where muscle of any thickness covers the part examined, it should be in a relaxed state, so as to facilitate as far as possible the close approximation of the pleximeter to the proper wall of the chest. The converse is the case when immediate percussion is employed; for the obvious reason, that a flaccid mass of muscle, in itself non-vibratile, must, besides, interfere with the transmission of sound from the subjacent parts.

106. It is scarcely necessary to insist upon the importance of observing, as far as possible, the same conditions, when percussing the two sides of the chest comparatively. Nor must it be forgotten, that in doubtful cases, the observation should be repeated many times and in various postures,—more especially, if the patient be in bed, the percussion should be performed several times from the right and left sides of the bed alternately. On the other hand, it is of essential consequence in some cases, for instance, in percussing the heart, that not only the posture

of the trunk be unchanged during examination, but that the limbs be kept perfectly quiet.

Healthy Standard of Pulmonary Percussion.

107. It is difficult, as has been said, to make intelligible by words the *quality* of the sound elicited from a healthy thorax; experience only can teach it. The *duration* and the *clearness* of the sound bear a definite relation to each other; whenever the former is considerable, the latter is proportionally marked, and *vice versâ.* And again, the clearness of the sound and the sense of resistance experienced by the fingers have a manifest connection: as the former increases, the latter decreases. Thus the sound is clearer in the infra-clavicular region than in the scapular; and so the sense of resistance is much less under the clavicle than upon the scapula. It is true there are a few exceptions to this law of relationship of the clearness of the percussion-sound to the resistance of the percussed surface; instead of invalidating, however, they strengthen the rule. Thus in the internal division of the clavicular and in the upper sternal regions, the sound is clearer than in others—for example, the infra-clavicular—where the resistance is less. This peculiarity manifestly depends upon the nature of the wall of the thorax in the former situations: being there wholly composed of bone, it cannot give way and rebound under percussion to the amount which the slight density of the subjacent parts would otherwise ensure; while, on the other hand, bone possesses a special resonance comparatively clear and high-pitched.

108. The properties of the percussion-sound of the healthy thorax vary materially in its different regions.

109. *Anterior Regions.*—It is necessary to assume a standard of comparison for the sounds in different localities, and that of the infra-clavicular regions may be used for this purpose. Here the sound is of considerable clearness, true pulmonary quality, and sufficiently prolonged to have a distinctly appreciable dura-

tion; while the parietal resistance is slight, and the elasticity marked. In the right mammary region, even at the upper edge, the clearness diminishes slightly on pretty firm percussion; while at and below the fourth interspace, though pulmonary resonance may be elicited by a very gentle tap, very perceptible dulness is caused, if the blow be firm, by the presence of the liver behind the shelving border of the lung: at the sternal edge of this region the heart in the majority of persons deadens the sound. In the inner part of the corresponding left region, the heart lessens the clearness of the sound and increases the resistance: this influence may be perceived more or less obviously as far outwards as the nipple. The entire of the right infra-mammary region is dull and resistant from the presence of the liver: the left lobe of this organ also renders the inner part of the left corresponding region dull; while the outer portion is similarly affected by the spleen, and the intermediate space gives either an amphoric and ringing or a tympanitic note from the subjacent stomach, if empty,—a dull one, if full. On deep inspiration a certain extent of lung encroaches in front of the liver superiorly, and may be discovered by very gentle percussion; even in expiration some lung-substance may be similarly detected in the left region, unless the stomach happens to be greatly distended.

Both clavicles towards their sternal ends give a peculiar sound of mixed pulmonary and osteal character, rendered somewhat tubular too by the immediate vicinity of the trachea; about the centre of the bone the tubular and osteal characters become less, the pulmonary more, marked,—whence an appearance of less clearness in the sound. Towards the humeral end of the bone the sound loses considerably in clearness. In the supra-clavicular regions externally, especially in females, the sound is quite as clear as in the infra-clavicular; clearer in the inner part, where a slightly ringing character is also elicited, unless the percussion be directed so as to avoid the trachea completely. The full resonance of this region may in moderately thin persons be detected even a little behind the anterior edge of the trapezius muscle. On the other hand, in persons free from thoracic disease, but either naturally very thin or emaciated from any disease, the outer part of this region often gives a wooden sound under percussion, and is very resistant; the first rib forms, as it were, the floor of the region, and its proper osteal resonance comes out modified by the intervening soft parts. There is sometimes a slight difference in the resonance on the two sides, on account of the apices of the lungs not rising to precisely the same height: but this follows no fixed rule.

The supra-sternal region gives distinctly tubular resonance, if the percussion be made at right angles with the surface; a mixture of osteal sound is detected, if the impulse be given even to a slight degree in a downward direction,—the proximity of the trachea and sternum explains this. In the upper sternal region the resonance is of the same mixed quality, as on the adjoining end of the clavicle, as far downwards as the second rib: here, at the point of convergence of the inner borders of the lungs, especially at the moment the organs are distended by inspiration, purer pulmonary sound may be elicited by gentle percussion. But occasionally, from excess of cellulo-fatty substance at the top of the mediastinum, the percussion-sound may be perfectly dull here,—a peculiarity which sometimes proves a source of serious clinical difficulty.*

From the third rib downwards the resonance is of complex character. The heart and liver with the intervening diaphragm give a dull sound, modified, however, by the peculiar bone-resonance of the sternum itself. If the stomach be in the condition to yield either an amphoric or a tympanitic note, one or other of these qualities may be detected, modifying still further the dull sound of the solid organs. This region further presents one of the most difficult practical problems in the art of percussion,—that of defining the line of union of the heart and liver. Now, although there are cases where to a practised ear and finger the differences in the pitch of the sound and resistance of the two organs will effectually mark out the contiguous edge of each of them, instances of the kind are rare. These characters failing, the line of demarcation may in many persons be traced by making gentle and forcible percussion alternately, while the patient holds his mouth widely open: the special amphoric quality of the stomach-note may then be detected between—and only between—these two organs; this I have repeatedly proved on the dead subject. But even this test will, sometimes from solid or fluid accumulation in the stomach—sometimes from unusual position of the viscera, wholly fail; the line can then only be found by uniting hypothetically the lowest point of the heart's impulse with the apex of the angle formed by the union of the upper free edge of the liver with the right free edge of the heart.

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* It may simulate, according to circumstances, the appearances of pericardial effusion, aneurism or tumor,—as will hereafter be fully shown.

110. *Posterior Regions.*—The sound yielded by the upper scapular region, though less clear than that of the infra-clavicular, is not, absolutely speaking, dull, nor altogether without pulmonary quality, even in fat persons; in very thin people the sound is sometimes very notably clear. The lower scapular region, while duller than the upper, and resisting to a marked degree the impulse of the fingers, has some faint pulmonary character in both respects; the spine of the bone, on mediate percussion especially, gives a clear osteal sound. The inter-scapular region holds an intermediate place in point of resonance between the upper and lower scapular.

The entire of both infra-scapular regions, especially in inspiration, gives a clear pulmonary note on gentle percussion as far as the eleventh interspace downwards. But forcible percussion on the right side brings out the dull hepatic sound from the lower edge of the region, as high as the ninth or even eighth rib, the amount of dulness gradually decreasing in the upward direction. On the left side the spleen, if large, or the stomach and intestines, modify the percussion-sound of the lung at the lower part of the region. Speaking roughly, it may be said that the extreme right base sounds somewhat duller, posteriorly, than the left. The influence of the kidneys, when of natural size, is practically null.

111. *Lateral Regions.*—The axillary region is highly resonant in its upper part, giving in many persons a sound clearer and of greater duration than the infra-clavicular region. On the right side the sound becomes dullish on strong percussion at the fourth interspace, and quite dull at the sixth rib, continuing so thenceforth to the lower border of the infra-axillary region. Below the sixth rib on the left side, though pulmonary resonance may still be elicited by gentle percussion, the spleen and stomach modify in their respective ways the quality of the sound.

112. Lastly, the sound elicited from the larynx and trachea (which is best obtained by filliping with the right middle finger one of the fingers of the left hand applied firmly to the surface, the patient's head being thrown back, and the tissues of the neck thus brought into a state of tension) is very clear, of considerable duration, and has a distinct hollowness in its quality,—is in fact tubular. These characters arise from the form of the tube and the quantity of cartilage in its walls. The resistance is greater under the fingers, in proportion to the clearness of the sound, than it would be in the chest.

113. The properties of the sound elicited by gentle and by forcible percussion differ to a slight degree in all regions of the chest. When strong percussion is used, the percussing fingers, by a sort of mechanical necessity, are allowed to rest on the surface for a moment; and thus a muffled character is given to the sound from interference with the vibrations of the chest-walls. Hence, force in the blow, instead of intensifying, weakens the sound and lessens its duration. Again, wherever an organ of greater density than the lung lies at some depth from the surface, the intervening space being occupied by pulmonary tissue, the sound will be rendered duller by striking

heavily, its duration diminished, and the sense of resistance increased: by employing force, the impulse is made to reach the deep-seated organ. For this reason a decrease in clearness may, as we have seen, be sooner detected by strong than by gentle percussion, in passing from above downwards in the right lateral and anterior regions; the influence of the liver is thus brought into play at a higher point of the chest. On the same principle the precise extent of the heart overlapped by the lung may be defined by alternately using some force, and by merely tapping the surface.

114. *Cæteris paribus*, the sound is clearer on the ribs than the intercostal spaces; this is especially true in thin persons,—the osteal sound mixes with the pulmonary, and raises the pitch of the resulting compound sound.

115. Numerous differences in the results of percussion of corresponding points of the two sides of the chest have been described, and traced to an obvious cause,—the presence of texture and organs of different densities in those spots. Variable thickness of the external soft parts will have a similar effect; thus the right infra-clavicular region is less resonant than the left in robust persons whose employment requires much use of the right arm: the pectoralis muscle enlarges from use. Here the explanation is clear; but the explanation is not clear when the right infra-clavicular region sounds duller than its fellow, in persons presenting no muscular thickening of this kind.* Such inferiority of clearness, however, the lungs being perfectly sound, is always when it exists, very slight: it holds good, whatever be the direction given to the percussing fingers, and whether they fall outwards towards the humerus or inwards towards the sternum.

116. The acts of inspiration and expiration modify the results of pulmonary percussion in three different manners: *a.* by altering the volume of the lungs; *b.* by altering their density; *c.* by altering the position of the heart and abdominal viscera.

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* Clinical Lectures, loc. cit. p. 196.

117. *a.* At the close of an ordinary expiration the right lung extends downwards as far as the sixth rib in front and the eighth laterally. The lower edge of the left organ sloping abruptly outwards and downwards from the middle line on the level of the fourth cartilage, again turns inwards a little, and then passes outwards, reaching laterally the eighth rib or interspace. From numerous trials on the dead subject, where no cause existed to modify materially the volume of the lungs, I infer that in expiration the lungs are from one to one and a half inches apart on the level of the second cartilage, and four and a half inches apart on the level of the fifth. From their lateral aspect the lower edges curve downwards and backwards, reaching the ninth interspace or tenth rib, especially close to the spine.

During full inspiration the lungs extend downwards in all directions somewhat further than the limits just mentioned; their inferior edge is then carried about an intercostal space and a half lower than after ordinary expiration, and proportionally still lower, when the lungs had been forcibly emptied of their air. At the same time the space on the left of the sternum, where, after expiration, the heart is in contact with the thoracic walls, becomes more or less filled with lung.

It is obvious, from these facts, that the *superficial extent* of surface, from which pulmonary percussion-sound may be elicited, will vary with the precise moment of the respiratory act at which the observation is made.

118. *b.* The density of the pulmonary tissue being in the inverse ratio of the air it contains, it is plain that the percussion-sound must be rendered duller in any given point of the chest by a full expiration. In other words, the sound yielded will vary in clearness according as percussion is performed at the close of inspiration or of expiration. The duration of the sound, and the sensation of elasticity perceived, vary, in the ordinary way, as the modifications of clearness.

119. *c.* The depression of the dull-sounding spleen and liver attending inspiration, replaced as they are by expanding lung,

tends during that act to increase the clearness of the percussion-sound in the lower regions of the thorax; the influence of the similar locomotion of the stomach and intestines will vary with the filled or empty state of those viscera. The heart pushed downwards and inwards, and receding somewhat from the chest-wall during inspiration, where its place is taken by the inflated lung, gives dulness of less extent and lower site than in expiration.

120. In consequence of this triple influence of the respiratory movements on the results of percussion, it is advisable, under all circumstances, and absolutely necessary in delicate cases, that the act of respiration be at the same stage of progress, when the two sides of the chest are percussed comparatively. The end of a full inspiration is in such instances the fittest moment for striking; as, by desiring the patient to hold his breath, we may then be certain of having both lungs in the same state for a short while. If the respiration be calm, however, such nicety is not required.

121. In the state of health the posture of the patient (except in so far as it may interfere with the act of striking on the part of the physician, or alter the tension of the patient's own muscles, or the relative position of the subcutaneous tissues,) does not *directly* affect the results of percussion of the surface, wherever this corresponds to lung-substance. In other words, ordinary changes of posture have no notable influence in modifying the relationship of the lungs and their containing walls to each other, or in altering the amount of air they contain, or are capable of containing. But as variation of attitude very sensibly alters the position of the heart, either in a downward, forward, or sideward direction, and also of the liver, the posture of the patient must always be taken into consideration in estimating the results of pulmonary percussion near the cardiac or hepatic regions.

Variations from the Standard of Pulmonary Percussion compatible with a Healthy State of the Thoracic Organs.

122. The sound yielded by the chest of different individuals varies in clearness; being, generally speaking, clear in proportion to the thinness of the walls. In accordance with this it becomes distinctly clearer in persons who, from a previous state of fatness, fall into one of emaciation.

123. The sound varies, too, with age. From the age of four or five to fifteen the pitch is high, the duration marked, the resistance slight; the walls are flexible, elastic, and commonly not much loaded with soft textures. The ossification of the cartilages, desiccation of the ligaments, and general stiffness of the thorax in old age impairs the freedom of vibration, and deadens the sound; and they would probably do so much more generally than is actually observed, were it not for the frequency of atrophy of the parenchyma of the lung in advanced life: wasting of the external soft parts, too, tends to the same modifying result.

124. Marked deformity of the chest, congenital or acquired, impairs the resonance over lung, healthy in itself. The existence of broken ribs, too, lessening freedom of motion on the affected side, and hence lessening the quantity of air in proportion to the pulmonary substance beneath, deadens the percussion-sound —a fact showing that slight dulness does not, after an injury of the kind, absolutely prove the existence of pulmonary congestion.

125. Unusual laxity of the ligamentous structures of the thorax renders the walls unfavourable to vibration. But, making all allowance for the various conditions referred to, there will still remain instances in which the chest is unusually clear or dull sounding, without its being possible to assign any satisfactory cause for the peculiarity. The real existence of such cases, however inexplicable at present, should always be borne in mind by the clinical observer.

PERCUSSION IN DISEASE.

126. Whatever be the nature of the morbid conditions of the sound discovered in any case, the space in which they exist may be either accurately defined or not; in other words, the morbid state, whatever it is, may either gradually pass into the healthy, or cease abruptly. In the latter case there is no difficulty experienced in ascertaining its precise limits; in the former, there often may be some indecision on the point, and it will be found useful to glide the pleximeter-finger rapidly over the entire region under examination, continuing the percussion all the time it is moved. In this way the exact line at which dulness or other change begins may be detected in very difficult cases.

§ ALTERATIONS OF SOUND.

I.—STATICAL SIGNS.

127. The statical changes in the percussion-sound, produced by disease, are comparatively few in number, and simple in nature, but the indications they furnish most precise and valuable. Concisely stated, these changes are :—

a. Diminution of clearness, gradually passing into perfect dulness,—the *duration* of the sound being proportionally *shortened*, and the *sense of resistance increased.*

b. Increase of clearness and of duration, with *decrease of resistance.*

c. Increase of clearness and of duration, with *increase of resistance.*

d. Alterations of quality.

e. Moveableness of dulness.

128. (*a*) Diminution of clearness, with its attendant changes, occurs wherever the density of the materials, underneath the part struck, is increased. Thus, deficient resonance exists, *first*, wherever any new material, of greater density than lung and air combined in the natural relationship, has accumulated within the

chest: whether it be in the lung proper, as in congestion, inflammation, abscess, gangrene, serous infiltration, apoplexy, simple chronic consolidation, infiltration or tuberous accumulation of exudation-matter, tubercle or cancer within the parenchyma;—or in the pleura, as in cases of hydro-thorax, and hydro-pneumo-thorax, hæmo-thorax, pleurisy in the periods of exudation, effusion, and retraction, empyema, serous infiltration of old false-membrane, and solid adventitious products of all varieties;—or in the bronchi, as in cases of abundant muco-purulent accumulation within the tubes;—or in the mediastina, as in cases of hypertrophy of their cellulo-fatty tissue, enlarged bronchial glands, abscesses, and adventitious solid products. *Secondly*, such deficiency arises where any condition, either physical or dynamic, exists, favouring expiration and impeding inspiration: as in obstructive diseases of the upper air-passages, spasm of the glottis, and spasmodic asthma,—in the latter affection the presence of pulmonary emphysema often prevents the failure of resonance from being discovered. *Thirdly*, dilatation and thickening of the bronchi, condensing the adjacent parenchyma, produce a similar effect. It is obvious, too, that serous infiltration of the parietes, abscesses, tumors, soft osteophytic enlargement of the ribs in chronic empyema (U. C. Mus. No. 4067), and thickening of the costal periosteum in syphilitic persons, will deaden the sound. The influence of the heart and great vessels will be elsewhere considered.

129. The characters of the dulness are not *per se* capable of revealing its cause, at least with very rare exceptions, in the present state of knowledge. Still the very intensity of the dulness and great resistance go far sometimes towards establishing the diagnosis in cases of intra-thoracic tumor. Between this extreme and the opposite one of slight impairment of sound in spasmodic asthma, all shades of difference may be observed.

130. (*b*) Increased clearness and duration of sound, with decreased resistance, or excess of elasticity, is noted, where the relative quantity of air within the chest is increased, but not

carried to such extremes as to interfere, by tension of the walls, with their vibration,—in what may be familiarly called the *rarefying* classes of diseases. Here may be enumerated pneumo-thorax, and hydro-pneumo-thorax, and atrophy, hypertrophy, and emphysema of the lung. It is possible that extreme anæmia, as originally suggested by Dr. Stokes, by lessening the relative quantity of blood in the lung, may increase the clearness of the percussion-sound.

131. (*c*) Increase of clearness and of duration of sound with increased resistance of walls, is observed where there is a surplus of air in the subjacent part, with considerable induration of tissue between the surface and the part containing that surplus, —a combination of conditions met with where a superficial cavity in the lung has a thin, indurated, and adherent external wall.

132. (*d*) As far as it can be rendered intelligible by words, the nature of the above three classes of alteration is explained by their names; the case is different with the fourth class, that of alterations of *quality* or *timbre*, which consequently require fuller description.

Instead of the quality *sui generis* which distinguishes the natural sound emitted by the chest, others, assimilable to those of various well-known tones, exist in certain states of disease. These morbid varieties of quality are referrible to three types: Wooden; Hollow; Tympanitic.

133. *The wooden* quality is very closely that of the sound yielded by mediate percussion of a common table, and distinctly conveys the idea of hardness. The duration of the sound having this quality is commonly less than in the natural state, and the sense of resistance experienced by the fingers is unusually great.

134. The *hollow* type occurs under three varieties,—the tubular, amphoric, and cracked-metal.

135. The *tubular* quality is precisely like that of the resonance emitted by the trachea under mediate percussion. The sound

it characterises is of some duration, and the thoracic elasticity somewhat impaired. This character is to be detected (always more readily if the patient's mouth be kept open) about the inner part of the infra-clavicular region in cases of pleuritic effusion, occupying the lower parts of the chest,—sometimes during the height of the disease,—sometimes, when absorption has just commenced; sometimes in the stage of retraction of the same disease. So, too, when solidified lung, pent-up collections of pus, or solid products of any kind lie between the large bronchi and the costal surface, the sound acquires this quality. Mere dilatation of the bronchi will have the same effect. Sound of this quality is most common in the infra-clavicular and upper mammary regions; but it may occur in the inter-scapular region from enlarged bronchial glands,* or over a great part of the back in cases of intra-thoracic tumor.† Excavations in the lung of small and medium size furnish similar resonance.

136. The *amphoric* quality, the tubular on a larger scale, is heard in typical perfection in the sound produced by filliping the cheek, when the mouth is closed and fully, but not too tensely inflated,—and also in the abdominal percussion-sound in certain states of distension of the intestines. I have observed this perverted quality over the entire upper front of the chest, including the clavicle and first bone of the sternum, in a few cases of pleuro-pneumonia, and in as great purity as it is ever heard over the intestines. When of this origin, the sound does not last long. But the common source of the sign is a cavity of large size, near the surface, and provided with hard and thin walls.

137. The *cracked-metal* quality (*bruit de pot fêlé* of Laennec) somewhat resembles that of the sound resulting from striking the back of the hands, loosely folded across each other, against the knee,—the contained air being forced out quickly and abundantly

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* Petrolini, U. C. H., Females, vol. v. p. 176. Here the tubular quality, distinct on gentle, disappeared on strong, percussion.

† Nature and Treatment of Cancer; case of S. C., p. 362.

between the fingers at each blow. When this character modifies the percussion-sound of the chest, there is coupled with it almost invariably a good deal of the amphoric quality, and the combination gives a result altogether *sui generis*, which, once heard, cannot easily be forgotten. Let there be a pulmonary cavity of large size with anfractuous walls, and communicating freely with the bronchi, the corresponding parietes being at the same time particularly yielding, and percussion will elicit this variety of quality. It is commonly said to depend on the collision of liquid and air produced by the blow; but the sudden propulsion of air, forcibly expelled from the cavity, against the walls of the passages with which it comes in contact, seems sufficient to generate it. In the first place, cracked-metal resonance is producible over cavities, when free from fluid,—at least if the absence of cavernous rhonchus is to be trusted to as evidence. In the second place, if, as I found several years ago,[*] the nose and mouth be tightly closed in a patient furnishing the cracked-metal sound when they are open, that character will at once cease to be producible, though percussion continues to give an amphoric note.[†] The fair interpretation of this fact seems to be, that the sudden rush of air from the cavity outwards, produced by the forcible blow upon the yielding parietes in the ordinary open state of the mouth and nose, but completely prevented by their closure (the only condition materially altered in the experiment), is the real cause of the phenomenon. Whether the communication with the external air be interrupted or not, the contents of the cavity may be dashed together by percussion: were the common notion correct, the cracked-metal character ought therefore to be elicited in both cases. This explanation derives support from the conditions of production of the amphoric and cracked-metal sounds by striking the hands, closed so as to form a hollow, against the knee: if they be so closed as to prevent air from being forced from between them by the blow, the amphoric character only is detected,—if air be allowed to escape freely, the character analogous to the cracked metal is superadded; yet here, certainly, there is no liquid to take part in its production. I have known a tubular note in pneumonia become distinctly cracked-metal when consolidation was at its height.

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[*] Lancette Française, 1834.

[†] The result will be the same, if the breath be purposely retained; a patient with tuberculous excavation, ascertaining this accidentally, amused himself with mystifying the bystanders, by sometimes having, sometimes not having, cracked-metal resonance.—Quin, U. C. H., Males, vol. vii. p. 291.

It has been well observed by Dr. Stokes, that a "metallic resonance, somewhat analogous to the cracked-jar sound of cavities, but evidently more diffused," is occasionally discoverable in cases of bronchitis, particularly in children. I have repeatedly observed this in infancy; it becomes especially likely to mislead, when there is evidence of tuberculous disease in extra-thoracic organs, as the brain or meninges.* The sound is certainly more diffused than, and otherwise different from, that dependent on excavation; but the absence of other cavernous phenomena, and the knowledge of the fact that bronchitis may simulate (for it does no more than this) the sound, are the best safeguards against error. The pliancy of the chest-walls explains the peculiarity.

If the chest of a crying infant be percussed in expiration, the resonance will be of cracked-metal quality, though the lungs are perfectly sound.†

138. The *tympanitic* character, as its name signifies, resembles that of the sounds of a drum. The note is very clear; its duration considerable; the resistance of the walls tense, drum-like, highly elastic.

It exists to the maximum degree in pneumo-thorax and hydro-pneumo-thorax, with considerable distension of the side; if, however, the distension be extreme, the amount of tympanitic quality lessens.* In emphysema, with bulging of the chest, it is also occasionally observed, but never in the perfection attained in pneumo-thorax. Further, as was first noticed by that accomplished observer, Dr. Graves, the quality of the note over pneumonic consolidation, sometimes, temporarily becomes tympanitic: of this I have now observed two positive examples at the right base, in one with, in the other without, plastic lymph on the pleural surface.

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* Wright, æt. 4; U. C. H., Females, vol. viii. p. 193.

† Bowen, æt. 4; U. C. H, Females vol. vii. p. 296.

139. (*e.*) The superficial extent and locality of percussion-dulness, important guides to the detection of its cause, will be dwelt upon in the description of individual diseases. It may be remarked here, however, that the area, within which loss of resonance is detected, may either be *fixed*, or *changeable with the position of the patient.* The former is by far the more common case; no matter how the posture be varied, the line of demarcation of the naturally and morbidly sounding parts commonly remains unaffected. But under certain comparatively rare circumstances, the boundaries of the dull sound may be more or less completely altered by causing the patient to vary his posture; the infra-scapular region, which may have sounded dull when he lay on his side, or reclined backwards, will give a clear sound, after he has remained leaning forward for a short while, and *vice versâ.* This movableness of the sign indicates movableness of its cause; and fluid collection in the pleura, from pleurisy, hydro-thorax, and perhaps hæmo-thorax, is the only physical state of which the percussion-signs have, hitherto, been admitted to be thus characterised: the fluid will, of course, gravitate to whatever part of the patient's chest his changes of position render the most dependent. It is, however, by no means so constant a sign of pleuritic affection as might, from *à priori* considerations, be expected. The liquid is retained *in situ* in some cases by adhesions, which easily explains the fixedness of the dull sound; in other instances, the explanation cannot be found.

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* Plympton, U. C. H., Males, vol. iv. p. 410.

I some time since met with a case in which a massy encephaloid tumor hung by a sort of peduncle of condensed lung-substance to the surface of the organ, and so loosely that it must have altered its position as the patient turned from side to side.* But there was enormous pleuritic effusion present on the patient's admission, which would of course have prevented the discovery of any change of place of the tumor, had the idea of looking for such locomotion occurred to me: the source of fallacy must be infinitely rare. More recently I ascertained, in a case originally seen by Dr. Evans of Birmingham, that even in cases of solid infiltration of a lung, decumbency on the sound side may displace the dulness of the diseased one,—carrying it to the opposite side of the median line at mid-sternum.

In hydro-pneumo-thorax the air and fluid may sometimes be made to change relative positions, to a certain extent, by changing the patient's posture; the site of the exaggerated resonance will, unless adhesions interfere, always be towards the upper surface.

140. The mechanism of tympanitic resonance in pneumo-thorax seems, on first thought, simple enough: the air-distended pleura and thoracic walls imitate pretty closely a common drum. Why, however, does the tympanitic quality become less marked, when air-distension reaches an extreme point,—when, in other words, the cause appears increased in amount? Whatever be the explanation, a similar phenomenon is observed in the drum; if a drum be tightened to the extreme point possible, and all escape of air from its cavity prevented, its sound, when struck, becomes muffled, toneless, almost null. Some maintain that the excessive stretching of the chest-walls or drum-ends impedes their vibration, in conformity with results obtained by Savart on the vibration of tense membranes; others, that the walls becoming vibratile when stretched to the maximum, their vibrations interfere with those of the contained air, and so nullify the sound, which is really and solely produced by that air. It may be that the mere size of the pleural sac has something to do with the phenomenon; an aperture is essential to sonorous vibration in large hollow instruments, whereas the vibrations of the air in small cavities are said to be soniferous, although perfectly closed: it is doubtful, however, that the

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* Dewing, U. C. H., Males, vol. v. p. 19. Museum, No. 4000.

difference in capacity of a moderately and a greatly distended pleural sac is sufficient to warrant this explanation.

141. The distinction of the amphoric and tympanitic qualities cannot be set aside practically, for they are absolutely different to the ear: the former, too, is suggestive of emptiness, the latter of fulness. Yet they are, in some cases at least, allied in mechanism. In the abdomen, for instance, a knot of intestine distended to the full with flatus will yield a sound of tympanitic quality; if the distension become less by the movement onward of some of the flatus, the sound will often instantly acquire an amphoric quality: judging from the absence or presence of gurgling sound, this holds true whether there be notable fluid or not in the spot. There are cases, too, in which the resonance partakes of both qualities.

I have never met with the amphoric percussion-note in general pneumo-thorax: but in a moribund patient of Dr. Garrod's (U. C. H., Ward 7), to whom I was called accidentally in the absence of that physician, and in whom I found, in addition to the more ordinary signs of copious pleuritic affection, *amphoric*, not tympanitic, resonance at the upper and inner part of the side, extending across the first bone of the sternum; the post-mortem examination, a few hours later, proved the presence of several cubic inches of gas in the site of the amphoric resonance. Local pneumo-thorax, at all events at the *upper* part of the chest, may then give an *amphoric* quality to the resonance: but the vicinity of the trachea and large bronchi probably exercised an influence on the quality, converting it from tympanitic into amphoric.

142. True tympanitic resonance is excessively rare over pneumonic consolidation; and I have never observed it in cases of pleuritic effusion. When occurring at the base of the chest, great distension of the stomach or colon might be suggested in explanation: but in point of fact the stomach and colon are rarely distended enough for the conversion of their common amphoric, into tympanitic, quality; and in the only two positive instances I have observed of pneumonic tympanitic sound, the consolidation was on the right side above the liver. Can the phenomenon depend on temporary secretion of air by the pleural sac? *

143. On the other hand, tubular, or the larger-scaled amphoric, quality is not very uncommon in acute hepatisation; is common in chronic solidification; and is an almost unfailing attendant on pleuritic effusion at some period or other of its course.† What is the cause of this? Formation of intra-pleural air, only in rare and exceptional cases, such as that just referred to [141]; for unquestionably, unless under the influence of gangrene, or special alteration

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* Vide art. Products, Adventitious; Cyc. of Anat. p. 145.

† My own experience would justify stronger language even than this: I have, in truth, *never* known pleuritic effusion run its course without tubular resonance, in cases where the opportunities for examination were sufficient.

of the fluids, such formation is not admissible on any existing pathological principle. Skoda's doctrine, that when the lung is moderately deprived of air, the sound becomes of the quality now under consideration, would refer the phenomenon to the partial expression of air from the substance of the lung by the pleural fluid or by the interstitial exudation, as the case may be. This doctrine will not explain the tubular quality so common at the apex in tuberculous consolidation, nor that attending certain large mediastinal tumors: neither is it accordant with the fact that in cases of effusion, where the resonance is highly amphoric near the sternum, it is commonly flatly dull in the axilla; the lung in the axilla must be compressed, as much as that near the sternum, by the effusion below. It seems to me plain, that the vicinity of the bifurcation of the trachea and large bronchus near the sternum has much to do with the quality,—a quality, be it remembered, imitable by filliping on the trachea: hence the original suggestion of Dr. Hudson, looking upon the tubular note as that of the bronchi conducted by solidified lung, has much reason in its favour. But, in turn, it is scarcely applicable to all cases, and much still remains to be learned on the point.

At the post-mortem examination of a case of acute tuberculisation and consolidation, the following singular facts were observed. "In the third and fourth right interspaces close to the sternum, where amphoric percussion-note was so marked at the last examination during life (forty-eight hours before death), the resonance is now totally without amphoric quality . . . . About eight ounces of serosity in the right pleural sac; entire right lung solid, bearing on its surface the impress of the ribs . . . . Corresponding to site of amphoric resonance in front, the lung is thoroughly solid and cuts almost crisply; this portion of the lung lies over the main bronchial tube, which, however, lies deeply . . . . At the *posterior* aspect of the lung, about three inches from the apex, close in towards the spine, and at about half an inch from the surface, is a cavity capable of lodging a good-sized walnut . . . Weight of lung fifty-six ounces." (Case of H. Hodson, U. C. H., Males, vol. ix., pp. 18—20.) The disappearance of the amphoric quality after death seems most mysterious: it had existed during the whole period the man was under observation, namely, from the 26th October to the 6th November. If we suppose that it disappeared between the last examination and death, this is equally inexplicable: there was nothing in the lung making it probable that a notable increase of consolidation had immediately preceded his decease.

II.—DYNAMIC SIGNS.

144. It has been seen, that in health the act of respiration modifies the results of percussion in three different ways; now in disease the natural modifications may be perverted or impeded, and hence a certain number of dynamic signs. Few of these

signs are of practical importance; but occasionally some of them prove valuable.

145. As regards the increased volume of the lung, and the consequent extension of pulmonary resonance, attending inspiration—neither will occur in cases of hydro-thorax, pleuritic effusion, and pneumo-thorax, in emphysema, and obstruction of the chief bronchus, either from foreign bodies or inspissated mucus within it, or from pressure, aneurismal, glandular, or other, without it. In a remarkable case of the latter kind, where the respiration was permanently weak, the failure of inspiration in increasing or extending the resonance of the side, contributed much to the diagnosis of obstruction of the main bronchus.* Further, in cases where, during expiration, air, which in health would escape, still stagnates within the chest, the residual and supplementary volumes are constantly on the increase [82], and there will not be a sufficient reduction, during expiration, of the area over which pulmonary resonance is discoverable.

146. *Mutatis mutandis*, the respiratory influence on the clearness of percussion may be similarly affected. In health, full inspiration increases the clearness of the sound of percussion, and equally so on both sides of the chest. Certain states of disease, impeding full pulmonary expansion on either side, interfere on that side with the production of the increased clearness discoverable on the other after full inspiration;—hence a sign founded on *comparatively deficient increase of clearness at the close of a full inspiration* on either side, the sound being equally clear on both in the ordinary state of respiration. Again, the sound of the healthy chest is somewhat deadened by full expiration, and equally so on both sides of the chest. Certain states of the lung destroy this equality by rendering the sound disproportionately dull in the situation where they exist: hence the sign of *comparatively great diminution of clearness at the close of full expiration.* Both these delicate signs sometimes give very satisfactory results in cases of small, irregularly

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* Mary Ransom; U. C. H., Nov. 1848.

scattered indurations, tuberculous or other, of either apex. The mechanism of the former is obvious; the latter depends on reduction of size of the lung, in expiration, bringing within a small space solid matter which had previously been more widely scattered.

Or, on the other hand, other states of the lung, by impeding the expulsion of air from the vesicles, render the sound disproportionately clear; hence the sign of *comparatively deficient diminution of clearness at the close of full expiration*, a sign discoverable in emphysema and obstructed bronchus. If there be air in the pleura, the same result will occur.

147. The cases in which respiration ceases through disease to exercise any influence on the position of the heart and abdominal viscera may be inferred from the foregoing exposition.

§ II.—ALTERATIONS OF RESISTANCE.

148. In his introductory observations upon physical diagnosis in general, Laennec makes a passing allusion to the "sense of elasticity perceived" in percussing; but in no instance refers to the diagnostic indications derivable from changes in this elasticity. Piorry and others have availed themselves of them fully, but their importance is not generally appreciated. There are cases of not very rare occurrence, in which erroneous inferences would almost inevitably be drawn from the sound elicited by percussion, were these not corrected by the information derived from the degree of resistance felt by the fingers. Take the case of a cavity seated close to the surface: the unnatural clearness of sound which sometimes distinctly exists over such cavities, quite independently of tubular or amphoric quality, might not only lead to an incorrect estimate of the state of the subjacent part, but also to the inference that the lung, in reality least affected, was the most diseased. The sensation of hardness and firm resistance experienced by the fingers at once discloses the true cause of the unusual clearness. Besides, the cases are

extremely numerous in which it is satisfactory to have the corroborative evidence, furnished by the state of resistance, in favour of the inference drawn from the sound. That doubt often exists as to the state of the sound on the two sides, is unquestionable; and in these cases the condition of the subjacent parts may frequently be settled by taking into consideration the amount of resistance. To those persons whose sense of touch is more delicate than that of hearing, this source of diagnosis is of especial value.

149. In describing changes of sound, a good deal has been incidentally said on those of resistance; but recapitulation is advisable. The resistance depends on the state of the contents, and of the walls, of the chest. All conditions lessening the relative quantity of air in the lung, while they deaden the sound, increase the resistance; they have already been enumerated. All conditions which conversely increase the relative quantity of air, decrease that resistance, while they increase the clearness of the sound; these also have been enumerated: pneumo-thorax is the typical disease. But the walls of the chest may be so stretched in that very affection by excessive accumulation of air, that the resistance becomes considerable, though the sound continues clear. Here the state of the walls themselves modifies the resistance; as it does likewise where solid or liquid matters accumulate in their substance, where the periosteum thickens, or the ribs either enlarge, or, as in the retraction-period of chronic pleurisy, become unduly approximated to each other.

SECTION V.—AUSCULTATION.

150. AUSCULTATION means the act of listening, and is termed pulmonary or cardiac, according as the sounds listened to are produced in the lungs, or in the heart.

151. The direct object of pulmonary auscultation is the appreciation of certain sounds audible on the external surface of the

chest, and either produced by the respiratory play of the lungs themselves, or transmitted in a modified form by these organs from others, as for example the heart, in which they are actually evolved.

152. The method of performing auscultation may be *immediate* or *mediate;* in the first case the ear is applied directly to the chest; in the second, an instrument of variable material and construction (originally a hollow cylinder of wood, to which the name of stethoscope[*] was given by Laennec, and has been retained for its modifications), is used as a conducting medium between the surface examined and the ear.

153. Both of these methods of auscultation have had their favourers and their detractors. The advocates of mediate auscultation urge that—The stethoscope can be closely applied to several points of the chest, as the axillary, upper scapular, supraclavicular regions, and acromial angle, where the ear cannot be placed in accurate contact with the surface;[†]—the use of the stethoscope enables the observer to auscult in a posture more easy to himself, than that he is obliged to assume if he apply the ear directly;—it is indelicate to place the head upon the persons of females;—it is disagreeable to bring the head in contact with the bodies and clothes of some of the lower orders;—the limits within which the various sounds are perceived are more nicely circumscribed with the stethoscope than the unassisted ear;—certain phenomena, as pectoriloquy, are more distinct when the stethoscope is employed. On the other hand, while the partisans of immediate auscultation admit that in lean persons it is difficult to place the ear appropriately in some few situations, they affirm that such cases very rarely occur, and that in all others the stethoscope is an *inutile lignum*,—the direct application of the ear giving as precise indications as the employment of that instrument, with less appearance of fuss and less real trouble. For my own part, I entertain no doubt

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[*] From *στηθος* "the chest," and *σκοπειν* "to examine."

[†] These points are still more numerous in malformed chests.

that Laennec and others have greatly exaggerated the superiority of mediate over immediate *pulmonary* auscultation in respect of the distinctness with which the phenomena are heard, and the precision with which they are circumscribed, in cases where both modes of auscultation can be employed; and that this distinctness and this precision are in fact greater in such cases with mediate or with immediate auscultation, according as the observer is more habituated to one or other of these modes of examination. It seems very plain, however, that as there are cases in which the ear cannot be directly applied, or in which it is disagreeable or indelicate to do so, mediate auscultation is the method with which the student should most closely familiarise himself; while, on the other hand, as it is often difficult to persuade children to allow the stethoscope to be applied, and as we may often desire to auscult an adult when no instrument is within reach, the ear should be practised in immediate auscultation also.[*]

154. The proper construction of the stethoscope has been a subject of constant dispute. As was felt by Laennec, theory, in the present state of acoustics, deposes in favour of the solid instrument; yet, as a hollow one is almost universally employed, it may be inferred that theory is somewhere at fault. From trials with hollow and solid ebony and cedar stethoscopes, I have come to the following conclusions:—That with the hollow

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[*] Beyond all doubt, first-rate skill in *pulmonary* auscultation might be acquired without using the stethoscope at all, though it is common to hear the *invention of the stethoscope* spoken of as constituting Laennec's claim to immortality. No! his name will be imperishable, because he *discovered auscultation*, (for the ideas of Hippocrates, Hook, and Double may without injustice be ignored,) described accurately the sounds it detects, and traced these sounds to their anatomical conditions. Persons, indeed, are to be found, who seem to think that the stethoscope possesses some mystic faculty of communicating diagnosis, and saving the auditor all trouble of thought; unfortunately, the instrument is no more than

. . . die todte Sprachrohr, das den Schall
Empfängt und wiedergiebt, und selbst nicht höret.

instrument the respiratory murmurs appear stronger and more open in quality,—with the solid, weaker and sharper, so much so that a bronchial character may be simulated; that cavernous phenomena lose in some measure their hollow quality with the solid instrument; that friction-sounds are sometimes materially better heard with this; that the natural vocal resonance over the trachea is hollower, graver, and better articulated with the hollow cylinder; and that the resonance of the observer's own voice, as he speaks, while auscultting the chest of another person (autophonia), is materially more intense with the solid than the hollow instrument.

Glass, gutta-percha, and metal have been tried, but are certainly inferior, for various reasons, to wood, cedar or ebony. The really important point is, that the ear-piece should fit the ear well,—it is as necessary to try on a new stethoscope, as to try on a new hat. Beyond this, all depends on the use to which the student puts the instrument, and not on the density of the wood or the direction of its fibres.

155. In performing auscultation, several precautions, affecting the observer and the observed, are to be attended to. 1. The chest should be uncovered; or if, from circumstances, such exposure be inadmissible, as thin a layer of clothes as possible allowed to remain between its surface and the stethoscope. 2. All friction between the stethoscope and the patient's or the observer's clothes should be carefully prevented. 3. The position of the patient should be regulated in the same manner as for the performance of inspection; an unconstrained state of the muscles being particularly necessary, in order to insure free entry of air into the lungs. The sitting posture is, every thing considered, the most conducive to perfect investigation, provided the chair employed have a tolerably high seat, and the observer be of the middle height; a tall person will find himself most at his ease, if the patient stands. It of course frequently happens that the recumbent posture is the only one the patient can easily assume,—he may be perfectly unable even to turn on either

side. Under these circumstances the utility of the flexible stethoscope has been insisted on, as it may be applied far back laterally, and even to the dorsal regions, if the body be inclined a little sidewards, without moving the patient.. I confess I have never yet seen an ordinary case of pulmonary disease, whether primary or secondary, in which, so long as it was a matter of importance to auscult the chest, the patient might not be raised sufficiently by careful attendants, to admit of the examination being efficiently made, for practical purposes, with the common stethoscope or ear alone. But attendants may not be within reach; and, in special cases, tendency to syncope may make it dangerous to raise the patient's head;—here the flexible instrument will be useful. If in the sitting posture, while the front of the chest is submitted to examination, the patient should sit not exactly erect, but with the trunk sloping a little backwards, the arms being allowed to hang loosely at the sides. When the observer proceeds to examine the lateral regions, the patient may be directed to clasp his hands on the top of the head,—in other respects, retaining his former posture; and, lastly, when the dorsal regions are examined, sit upon the chair sidewise or, if a male, astraddle, with his back to the observer, his arms crossed, and his head bent somewhat forwards. *Mutatis mutandis*, the same precautions are to be taken when the patient stands, lies, or sits up in bed. 4. It is of importance to apply the stethoscope firmly, but not forcibly, to the surface: too slight or too strong pressure interferes with the accurate transmission, or alters the character, of the sounds. 5. Great care must be taken to insure accuracy of contact between the skin and every point of the circumference of the end of the stethoscope; as a necessary condition for this, the instrument must be applied perpendicularly to the surface, and held until firmly placed, by its applied end: the auscultator may then readily assure himself with the fingers, whether the skin and the edge of the instrument are in accurate apposition. 6. The posture of the observer should be free from all constraint; he should apply his ear to the stethoscope with as much care, as the instrument to the chest; concentrate his attention upon the sound examined; and, unless he be thoroughly experienced, proceed, as far as is compatible with the patient's safety, slowly with his examination. The motto *festina lente* is a good one for the beginner in the study of physical diagnosis. 7. It is advisable to commence the auscultation of patients, while they breathe in the manner to which they are naturally inclined; because it is important to ascertain the precise condition of the *tidal* respiration, and because directions for the regulation of the act often puzzle. Some individuals, however, absolutely require guidance: as the moment they perceive the instrument applied to their chest, they throw the muscles of the trunk into violent and unnatural motions, which materially impede the entry of air into the lungs. The simplest way of making such persons breathe in an efficient manner is, to perform several quick noiseless respirations before them, and desire them to imitate these. This method will, however, occasionally fail; our object may then be gained by desiring them to sigh, to speak, or to cough. The deep inspiration required for the performance of these acts will at once enable the observer to ascertain the condition of the sounds; and, indeed, there are many states of the lung in which, quite irrespectively of the patient's manner of breathing, much information may be gained by a single cough. It is to be remembered, however, that by these artifices we lose the simple tidal respiration. 8. The sounds produced in the pharynx by the passage of the air are liable to be confounded with the true pulmonary sounds of respiration; the error may be avoided by directing the patient to open the mouth, if it have been previously shut, and *vice versâ*. If the sounds heard have their seat in the lungs, they will suffer no notable change from this opening or closing of the mouth; if in the pharynx, they will be more or less modified in character. It is, however, only in unusual cases that a direction of this kind is necessary to enable a practised auscultator to avoid the error referred to; though it may often be advisable to corroborate thus the impression derived from ordinary examination. The sensation of distant production which attends the pharyngeal murmurs, and the occurrence of a distinct interval of time between inspiration and expiration (a point to which I particularly recommend attention), will suffice to distinguish them from the true pulmonary sounds. With ordinary watchfulness, the observer may distinguish the two kinds of sound at the same time. 9. Both sides of the chest must be submitted to precisely the same examination,—conducted precisely in the same way,—as already explained in reference to percussion. 10. Auscultation should never be considered complete, until the entire chest has been examined: it is often in some other situation, where the symptoms would least have taught us to look for disease, that auscultation proves its existence. 11. In acute affections, auscultation should be repeated twice, at least, in twenty-four hours. 12. The student should accustom himself to the use of both ears.

§ I.—The Sounds of Respiration.

I.—IN HEALTH.

156. Two sounds, discoverable by auscultation of the breathing-apparatus in the state of health, attend each act of normal respiration; one, corresponding to the movement of inspiration, the other, to that of expiration. These are the *inspiratory* and *expiratory sounds.*

157. The *essential or primary properties* of these sounds, practically considered,—those which, in their modified states especially, possess diagnostic importance,—are: *Special Character and Quality; Pitch; Intensity; Duration; Liquidness; Softness; and Rhythm.*

158. By the *special character* of a sound is understood that peculiarity which must, under all conditions of intensity, duration, rhythm, pitch and key, distinguish it from others; the special character of the sounds of a piano-forte, for example, will

invariably differ from that of the tones of a violin. Here also, for the purpose of simplifying the object as far as possible, without incurring any material sacrifice of accuracy, may be included that property of sound known as *quality;* though, in point of fact, the *quality* or "timbre" of a sound is a different thing, acoustically speaking, from its *character.* Thus, two voices, identical in point of character and of register—say, tenor —will sound the *same note*, in the same *rhythm*, with the same amount of *liquidness*, with the same *intensity*, and for the same *duration* of time, and yet a marked difference shall exist in the sensations impressed upon the ear by the two tones: that difference depends on the *quality* of each; and by it any two tenor or soprano voices are as easily distinguishable from each other, as any one of the four from those of bass or contralto register. Variety in quality is supposed to depend on varying form of the sonorous waves; but the conditions determining that form have not been ascertained. As the quality of the notes of musical instruments varies with the precise form and properties of the material composing them (the secret of the Cremona violins lay in their form and component woods), it may reasonably be conjectured that the form of the vocal tube and the tension, dryness, elasticity, and other properties of the tissues engaged in the production of laryngeal and pulmonary sounds modify the quality of these.* The *pitch* of sounds rises as the frequency of the vibrations in a given time of the sonorous body; the evident variations in pitch of the respiratory sounds under different circumstances immediately depend on variations in that frequency,—but why, or through what mechanism, the frequency is affected by different anatomical conditions, is unknown.

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* Some of the conditions regulating the quality of the human voice are theoretically under the influence of the will; and, as matter of experience, it seems certain that effort—and imitative effort especially—will modify the quality of singing tones. Thus, I knew a tenor singer who managed during the opera-season to throw into his voice, one of every-day tenor character, a something of the marvellous quality of Mario. This person assured me, that the power, which almost completely disappeared with the annual disappearance of his model, came of imitative effort.

159. The terms *intensity* and *duration* explain themselves. The notions of *dryness* and *liquidness* of sound may be at once obtained by squeezing close to the ear first a perfectly dry, and then a moistened sponge. Similarly, if we press together a mass of wool held beside the ear, the property of *softness* in sound will at once become intelligible; its converse, *hardness*, by grating together any two hard bodies. The *rhythm* of a sound means its mode of progression or evolution, which may be continuous and equable, or interrupted and jerking.

160. Although in originally establishing the varieties of morbid breathing, every respiration-sound requires to be analysed in respect of these various properties, the complexity of the matter is much less in actual practice than it seems; for experience proves that several of these properties are almost invariably altered simultaneously, and of course such *compound states* may be described for convenience sake by single phrases.

161. The properties of the sounds differ in the various divisions of the respiratory organs: for each of these divisions there is a healthy type of respiration, termed, *pulmonary; bronchial; tracheal; laryngeal; pharyngeal;* according to the part of the respiratory passages from which the sounds audible externally are transmitted.

162. The sole point in which these varieties of respiration agree is, that in all of them the audible sound may be resolved into two—an inspiratory and an expiratory. From their numerous distinctions they require separate consideration.

163. *Pulmonary.*—The pulmonary *inspiratory* sound is of gentle breezy character; neither liquid nor dry; soft; of a certain intensity and duration; and in respect of rhythm, gradually developed and continuous. The *expiratory* sound, slightly harsher and hollower than its predecessor, and of lower pitch, is about three or four times the weaker and shorter of the

two, and in some persons, especially at the left side of the chest, is actually inaudible.*

164. The term "breezy" seems the fittest by which to describe the *character* of the healthy respiratory sounds. They suggest in their pure state the sighing murmur of the breeze among leaves, the only difference being one of intensity. The use of the term *vesicular*, in speaking of the natural condition of these sounds, has led to an erroneous impression. It was originally applied to designate the seat of their production; but not a few persons have incorrectly referred the term to their special character. There is nothing in the nature of the respiratory sounds suggestive of a connection with vesicles; and, whenever such character occurs, the phenomenon it attends is morbid.

165. The two sounds so closely follow each other in each healthy respiration, that they may, practically speaking, be said to be continuous. This continuousness, indeed, forms an important character of *pulmonary* respiration of *healthy* type. It would of itself be sufficient to announce the lung as the part ausculted; for it will be found that in proportion as auscultation is practised, in health, at a further point from the pulmonary parenchyma, so will the two sounds be more and more distinctly

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* The fact that the escape of air from the lungs during expiration is commonly attended with audible sound was known to, and is distinctly stated by, Laennec. But the importance of the expiratory murmur, the valuable indications its modifications afford in the diagnosis of disease, did not sufficiently attract his attention; and to the late Dr. Jackson, jun., of Boston, U.S., belongs the credit of conceiving the value and extent of information which might be obtained from its analysis. In a most ingenious paper, read in 1832, before the Medical Society of Observation of Paris, that zealous inquirer forcibly drew attention to the subject. M. Louis and several of his pupils submitted the remarks of Jackson to the test of observation; his announcements were found generally correct, and thenceforth the separate consideration of the expiratory sound became with them habitual. Dr. Cowan subsequently favoured the English public with a valuable paper on the subject ("Lon. Med. Gaz." vol. xviii. p. 332). M. Fournet has since investigated it more thoroughly than his predecessors, and has popularised what had previously been known only to the comparatively few; but he seems to have fallen into repeated errors of over-refinement.

eparated from each other by an appreciable interval of time,—n interval which consequently attains its maximum opposite he larynx and upper part of the throat.

166. What is the site of production of the pulmonary sounds?—and what is the mechanism by which they are generated? M. Beau * endeavoured to show that the respiratory sounds, heard on the surface of the chest, are not produced in the lungs; but are simply the pharyngeal sounds, attending the entrance nd exit of air through the pharynx, transmitted through those rgans. Two facts seem to disprove this theory;—the existence f an appreciable pause between the respiration-sounds opposite he pharynx, while there is none such over the pulmonary structure; and the occasional possibility of hearing at one and the same spot of the chest-surface, both ordinary respiratory, and pharyngeal, sounds. Besides, the two pharyngeal sounds re quasi-equal in length, the pulmonary extremely unequal; nd destruction of portions of the fauces does not alter or impair he pulmonary sounds. The three first of these objections are equally fatal to Dr. Spittal's theory,† that in the glottis is the main origin of the sounds. Besides, there is no direct ratio between the intensity of sound heard over the larynx and that audible over the chest: the loud pulmonary inspiration-sound with which air makes its way into the lungs, through an rtificial opening in the trachea, is well known;‡ while, *per contra*, laryngeal and tracheal stridulous respiration is commonly attended with very weak pulmonary sound. Laennec believed that the sounds were engendered by "the entrance of the air into, and its expulsion from, the air-cells of the lungs;" and this, with a qualification to be presently considered, appears to be their essential cause. The terminal portions of the bronchi are also probably concerned in the sounds,—the vibrations of the moving air, and moving parenchyma, and the distension

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* Arch. Gén. de Médecine, 1834.

† Edin. Med. and Surg. Journal, vol. xli.

‡ Cole, ætat. 4; U. C. H., Females, vol. ix. p. 167.

of the air-cells, all, doubtless, contributing to the general result. The excess of inspiratory over expiratory sound seems explicable by the greater resistance of the textures during inspiration, and by the current being in the former instance directed towards, in the latter from, the ear of the observer. If a sponge, slightly moistened, be placed between two stethoscopes, and one person breathe naturally into one of these, while a second auscults by the other, it will be found that the difference in force of the sonorous currents towards and from the ear of the auscultator is about the same as between the intensities of natural inspiration and expiration. And, again, if a sponge in the same state be held close to the ear, and alternately pressed and allowed to expand, it will be found that the closure of the cells of the sponge is almost noiseless, their expansion accompanied with well-marked sound: the ratio is very much that of the expiratory and inspiratory sounds.

But Laennec was incorrect, I feel persuaded, in supposing that the actual air of each inspiration is directly concerned in generating the corresponding sound. The tidal air, in truth, does not reach the cells. In calm breathing the stagnant air [74] is never expelled from these: in forced breathing the supplementary share of the stagnant volume is expelled; but the residual and persistent quotas remain. Clearly, then, it is not the tidal air itself that, impinging against the cell-walls, evolves the sound; but rather the supplementary and residual volumes which, in their retreat before the influx of tidal air, recover the position they had partially evacuated in expiration.

167. The conditions of the pulmonary murmurs may vary within certain limits from those just described, without the type of respiration ceasing to be compatible with health of the lungs. In other words, there are *healthy varieties* of respiration. They are referrible to the following circumstances: *age; sex; the part of the chest furnishing the sounds; the rapidity and fulness of respiration; temperament; and idiosyncrasy.*

168. *Age.*—The description given of healthy respiration refers

to that of adults; at either extreme of life its characters are different. In infancy the *intensity* of the sounds is considerably greater than at a more advanced age, all the other properties of both sounds remaining unaltered both positively and relatively (*puerile* respiration); the expiration-sound appears disproportionately long only from its greater intensity. The smallness of the vesicles at this age, and hence the more extensive surface, concerned in the production of the sounds, seems to account best for their excess in infancy. The slightly greater frequency of respiration in childhood seems inadequate to its explanation, especially as there is no such difference in the laryngeal respiration of childhood and adult age. In old age, on the other hand, the *intensity* of the sounds is sometimes diminished, and the duration of inspiration lessened, while that of expiration is increased (*senile* respiration). Healthy senile respiration, as it may be called, differs from morbidly *weak* respiration in the increased duration of expiration. Andral has referred to its characters, and justly connects it with the rarefied and inelastic state of the lung which arises, as a condition of natural decay, in advanced life.

169. *Sex.*—The pulmonary respiration-sounds are generally louder in the female than the male; and certain peculiarities, to be presently mentioned, are of more frequent occurrence in the former than the latter.

170. *Region and Side of the Chest.*—The sounds are fuller superiorly than inferiorly, especially in women, and in front than behind. They are audible even at the lower edge of the right and left infra-mammary regions on full breathing. Between the scapulæ, and over the upper part of the sternum, the respiration presents more or less distinctly a bronchial character.

The characters of the inspiration-sound do not differ in the corresponding points of the two sides of the chest to any appreciable amount: and there is no excess, as a constant condition, in the duration and intensity of the expiration-sound in any one part of either side as compared with its fellow spot; but in a certain proportion of persons, especially females, as originally shown by M. Louis, the expiratory murmur is longer and louder in the supra and infra-clavicular, the supra-spinata, and the inter-scapular regions on the right side than the left. The nearer the inner part of these regions the comparison is made, the more marked the difference: while very notable near the sternum, it may be imperceptible at the acromial angles. The excess of expiration signifies an approach to bronchial character.

171. *Rapidity and fulness of Respiration.*—The *intensity* of the respiratory sounds increases directly as the rapidity of breathing: their *duration*, as its fulness. When the respiration is at once full and rapid, both those properties are affected simultaneously.

172. *Temperament.*—The respiration-sounds are, generally speaking, of greater intensity in persons of nervous temperament, or labouring under certain nervous affections, as hysteria, than in persons otherwise constituted.

173. *Idiosyncrasy.*—In some individuals the respiratory murmurs are unusually weak or unusually strong,—and the peculiarity, not being traceable to any particular cause, is spoken of as an idiosyncrasy.

The proportional duration and intensity of the expiratory sound vary much in different chests; in not a few persons expiration is completely unattended with audible sound. This absence of expiratory murmur is, according to my experience, most frequent in males; and when it exists, is a natural peculiarity, and in no instance the effect of disease. The exception which occurs to this latter statement, in some cases of emphysema, is only an apparent one.

174. *Bronchial respiration*, audible between the scapulæ, and at the upper end of the sternum, in the sites corresponding to the bifurcation of the trachea, wants in both its divisions the perfect softness and gentle breeziness belonging to the pulmonary species; both are slightly harsher, of higher pitch, more rapidly evolved, especially the expiratory, and follow each other less

closely, are less accurately continuous, than in pulmonary respiration. In some persons the respiration is very purely pulmonary even between the scapulæ.

175. *Tracheal, laryngeal, pharyngeal respiration.*—The respiratory sounds, as heard over the trachea, larynx, and pharynx, are considerably more intense, less soft, drier, hollower in quality, more whiffing, of higher pitch, and more rapidly evolved, though of greater duration, than in the divisions hitherto considered. While in pulmonary respiration the two sounds differ very materially in duration and intensity, in these upper sections of the system they tend to equalisation in these respects,—the second perhaps slightly exceeding the first; and instead of being almost continuous, are separated by an interval of some duration.

II.—IN DISEASE.

176. The phenomena discoverable by auscultation of the lungs in disease are:—Modified breathing-sounds; and Adventitious sounds produced by the act of respiration.

A.—*Modified Respiration-Sounds.*

177. It is extremely rare to find one only of the primary properties of the respiratory sounds affected; in by far the greater number of cases two or more of them suffer alteration at the same time; and thus are produced compound conditions of change, which may be described as distinct species of morbid respiration, and classified as follows:—

Species of unhealthy respiration distinguished by changes of—

Duration and *Intensity ;* exaggerated, weak, and suppressed respiration :

Rhythm, either solely or in conjunction with other properties; jerking, and divided respiration; deferred inspiration; unfinished inspiration; altered ratio of inspiration to expiration :

Quality, and in addition other properties; harsh and bronchial respiration, and blowing respiration, with its main varieties, simple and hollow.

178. *Exaggerated respiration* is essentially distinguished by an increase in the intensity and duration of both sounds, *especially the expiratory*,—an increase unattended with modification of any kind, either in respect of special character, softness, or liquidness. It is, likewise, termed *supplementary*, because the pulmonary tissue, in which it is produced, often supplies by increased energy the loss sustained by the inactivity of some other part; and *puerile*, on account of its similarity to the natural respiration of children. However, there is this difference between the supplementary respiration of the adult and the natural respiration of infancy, that in the former the expiratory sound is proportionally more affected. Undue prolongation of the expiratory murmur depending directly on disease, may be distinguished from that of exaggerated respiration, by its being attended with modifications affecting the *quality*, *softness*, and *liquidness* of the murmur.

The causes of exaggerated respiration are unnaturally rapid influx and efflux of tidal air through the lung, together with an increase in the quantity and the force of impulsion of the retreating residual air [166] against the walls of the vesicles, and probably, in the *number of these expanded by each inspiration* in the spot;—all this being the result, except in very rare cases, of the inaction of some part of the same or of the other lung. Of very variable seat and extent, it occurs: (1). In healthy tissue, adjoining parts *obstructed* by bronchitis, new productions or foreign bodies in the bronchi; *condensed* by plastic effusion, by pneumonia, by tuberculous, cancerous, or simple consolidation, by apoplectic effusion, &c.; or, *rarefied* by vesicular emphysema: (2). In healthy tissue, suddenly *released from bronchial spasm*, as in spasmodic asthma: (3). In tissue itself affected with *hypertrophy*. Exaggerated respiration, then, though not a *direct* result or sign of disease, except in the last rather doubtful case, furnishes valuable *indirect* evidence of its existence, and bears the same relation to the cause producing it, as, to use the happy expression of Fournet, the shadow to the substance. The closer the examination is made to the actually diseased part, the more intensely will the exaggerated respiration be found to be developed.

179. *Weak respiration* is characterised by a simple diminution in the *intensity* and *duration* of the respiratory sounds, without change of their other properties. When the quality of the respiration is simultaneously altered, the breathing belongs to another type. There are two varieties of weak respiration; the *superficial* and the *deep-seated.* In the former the weak sounds appear to be produced at the surface of the lung or immediately under the walls of the chest; in the latter, at a greater or less distance from these walls. The physical conditions present explain these peculiarities: in the latter, the portion of lung in which the weakness exists is in reality removed to a certain distance from the walls of the chest by the intervention of adventitious fluid or solid matter; in the former, no such removal of the pulmonary substance occurs.

Weak respiration depends on deficient flux and reflux of air in the affected part, itself commonly caused by mechanical obstruction. It is of variable seat,—limited to a spot in one lung, or pervading the entire of both lungs.

The *superficial* variety occurs, in a *persistent* form, in obstructive diseases of the larynx and some of the pharynx; narrowing or obliteration of a bronchus by contraction, thickening of mucous membrane, accumulated mucus, or pressure of tumours, cancerous or other; bronchitis; chronic consolidation of the lung, or infiltration with tubercle or other morbid product in a limited space; vesicular emphysema; pneumonia, previous to engorgement and after resolution; pulmonary œdema; pulmonary apoplexy, and imperfect respiratory movements from paralysis. In an *intermittent* form, it occurs in pleurodynia, in the dry and plastic stages of pleurisy, spasm of the glottis, spasmodic asthma, and where foreign bodies block up the air-passages. This intermittence constitutes an important feature in the physical signs of a foreign body in either bronchus, depending manifestly upon its motions from place to place, and consequent greater or less interference from time to time with the entry of the air. In some cases of pneumonia the same intermittent character of respiration may be observed; it indicates intermittent pressure on the main bronchus of the affected side.*

The *deep-seated* variety occurs in the effusion-period of pleurisy, in hydrothorax of moderate amount, and in pneumothorax.

In Cholera Asiatica the weakness of respiration seems partly dynamic, and partly of hæmic, or blood, origin.

180. *Suppressed Respiration* consists in a total absence of the respiratory sounds, without their being replaced by any kind of audible phenomenon. There is a complete negation of breathing-sound in the part; but, it is urged by M. Fournet, there is still heard in some cases, towards the close of the movement of inspiration, a slight noise, "which appears to result from the lateral pressure, exercised on the pulmonary tissue, of the column of air which fails in entering the bronchial ramifications." For this noise he proposes the name of "sound of pulmonary compression." I have not, in such cases, been conscious of the recurrence of a distinct sound; but an indefinable sensation of struggle within the chest against obstruction may often be perceived.

The causes which produce weakness of respiration entail, when carried to extremes, its total suppression. It is probable that the respiratory sounds are actually more or less extensively suppressed in almost all cases of infiltrations of the lung, tuberculous or other; but it is, for obvious reasons, in rare instances only that the suppression can be detected,—adjoining exaggerated respiration masks the local deficiency. In some rare cases of pneumonia, independently of accidental coexisting obstruction of a main bronchus, there may be no audible respiration,—I have seen two such; but, clinically, the common causes of such suppression are fluid and aeriform effusions in the pleura, and complete obstruction of a bronchus; in some rare cases of vesicular emphysema, and during intense paroxysms of spasmodic asthma, all respiration-sound may be locally deficient.

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* Beckett, U. C. H., Males, vol. v. p. 231.

181. *Jerking Respiration.*—When the movement of inspiration, instead of being accompanied by a murmur continuous from the outset to the close (which may be represented thus ......), is attended with a sound of an interrupted character, divided into several unequal parts, (thus ... | .. | .... | ... &c.,) the respiration may be called *jerking*. The expiratory sound more rarely possesses this peculiarity, and, as far I have observed, is scarcely ever affected alone; but it is generally somewhat increased in duration, while the inspiratory (if the moments, during which sound is actually perceptible, be alone considered) may be somewhat decreased in this respect.

Jerking respiration may exist, through the entire of one or of both lungs, when it deserves the name of *general;* this is very rare: or be limited to a certain spot, when it may be called *partial.* The former variety is observed in incipient pleurisy, pleurodynia, pulmonary and intercostal neuralgia, hemiplegia, spinal irritation, hysteria, and spasmodic affections of the air-passages. The partial variety occurs in cases of solid lung-infiltration and of pleuritic adhesion.

182. *Divided Respiration.* Instead of the two sounds, inspiratory and expiratory, succeeding each other so closely in each act of respiration that they may be considered continuous, they are sometimes separated by a distinct interval or pause: to respiration of this rhythm the name of *divided* may be given. In advanced emphysema this separation of the sounds clearly appears to depend on the destruction of the walls of the air-cells, and the impaired elasticity of the remaining texture. In all the varieties of bronchial and blowing respiration there is a pause, more or less marked, between the two sounds.

183. *Deferred Inspiration.* In emphysema each inspiratory action of the chest often commences, and continues for some seconds, before sound is produced: the closing instants of the

act only are attended with audible sound. The sound of inspiration may be said to be deferred.

184. *Unfinished Inspiration.* Conversely, the inspiratory sound sometimes ceases, before the accompanying expansion of the chest: this defect may be observed in consolidations of various kinds.

185. *Altered Ratio of Inspiration to Expiration.* Instead of the former of these sounds being to the latter very closely, as 3 : 1, in point of intensity and duration, the ratio may be converted into as 1 : 4. This enormous relative prolongation of expiration is only met with in emphysema; but minor degrees occur in various indurated conditions of the lungs, and in pleuritic effusion and pneumo-thorax. I can scarcely believe that prolongation of the expiratory sound is capable of existing independently of other alterations, as I have never met with it to any well-marked amount without co-existing change of quality. However, in some cases, the increase of duration is much more striking, and therefore, clinically, more important than the change of quality;—occasionally in tuberculisation, often in emphysema.

The signification of the expiratory sound was, in some degree, mistaken by its re-discoverer, Jackson; he exaggerated its specific importance as a diagnostic sign of tubercles, and in this he has been very generally followed. Many persons forget that what may appear in a given individual, as compared with another, prolonged expiration, is really in him a natural state; some confound with it the pharyngeal expiratory sound; the normal existence of lengthened expiratory sound at the right apex of many females is habitually forgotten; and too few observers seem to be aware that, under whatever circumstances an obstruction exists to the free circulation of air in the lungs, the expiration will be prolonged,—an obstruction which rarely, if ever, acts on the duration of the sound, without affecting some of its other properties. The fact of obstruction appears to me to account satisfactorily for the phenomenon. Expiration is, unless in exceptional cases, sonorous under all circumstances: where the reflux of the tidal air is rendered more difficult and slower than natural, the most natural consequence in the world is, that the attendant sound should be proportionally intensified and lengthened.

Diminished elasticity of the lung will, if there be obstruction superadded, produce the maximum prolongation, as in emphysema with bronchitis; very often, however, the sound spoken of as prolonged expiration in this disease is nothing more than sibilant rhonchus,—true breathing-sound being totally absent.

186. In some cases of incipient tuberculisation the tidal air seems to struggle against minute obstructions in the finer tubes, whence a rhythm of sound resembling that of a *cogged wheel* in rotation: it appears probable that glutinous mucus adherent to the walls, and which the air, in its flux and reflux, disturbs, without forming into bubbles, may be the cause of the phenomenon; the nature of the sound is rhonchoid, and it seems to pass into actual rhonchus in some cases.*

This peculiar rhythm is most commonly found in connection with tubercle; but I have observed it at one or both apices, when free from solidification of any kind.† It may be most marked with expiration, and lose in distinctness in forced breathing.‡ It is often found at a consolidated apex, when fine liquid rhonchus exists at the base.§

187. To the present head, also, belongs in part, the *intermittent* weak respiration, described a moment since.

188. In *harsh respiration* both sounds have lost their natural *softness;* a peculiar *dryness* accompanies them; the breezy character of health is exchanged for one sharper and more

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* M. Kenny, U. C. H., Females, vol. vii. p. 9.

† R. Smith (subcutaneous cancer), U. C. H., Males, vol. vii. p. 876, or Med. Times, 1852.

‡ Tagg, U. C. H., Females, vol. ix. p. 83.

§ Simmons, U. C. H., Males, vol. ix. p. 57.

blowing, which is generally more marked in expiration than inspiration. The *intensity* of the expiratory sound appears augmented from this superadded character; its *duration* is increased: both these latter properties may be, and commonly are, unaffected in the inspiratory sound. This type insensibly passes into the higher grade—*bronchial respiration.* Both sounds are now *rough* and *hard,* and notably more *dry* than in the natural state; the sharp blowing quality is heard in inspiration as distinctly as in expiration, and in the latter to a greater degree than in respiration of the previous type. The *intensity* of both sounds appears increased, the *duration* of expiration is considerably augmented, and is even greater than in normal bronchial respiration: otherwise morbid breathing-sounds of this type closely resemble the natural sounds in the larger bronchial tubes.

In harsh respiration the expiratory sound commonly alone suffers change of quality; in the bronchial variety the inspiratory shares in the alteration. The usual earlier implication of the sound of expiration was first noticed by Jackson, and has since been insisted on by M. Fournet. This writer states that alterations in quality always set in with the expiratory sound, and only affect the inspiratory secondarily. With this statement I find that my own experience accords in respect of chronic maladies, if the word habitually be substituted for always: I believe it to be correct also in the very great majority of cases of acute alteration of the parenchyma of the lungs. There is, however, a kind of rare bronchial respiration in which the inspiration alone suffers, the expiration retaining its natural properties; this seems to me insignificant in diagnosis, and rather to be an individual peculiarity of healthy breathing.

Harsh respiration, attending condensation or rarefaction of pulmonary substance, and dryness of the mucous membrane of the bronchi, is observed in incipient tuberculisation, dry bronchitis, vesicular emphysema, chronic pulmonary consolidation, dilatation of the bronchi, and incipient cancerous infiltration of the lung,—in cases where the lungs are slightly compressed by plastic or tuberculous matter in the pleura,—in the resolution-stage of pneumonia,—at different periods of pleurisy and in pulmonary apoplexy. Bronchial respiration is met with under all circumstances of slight condensation of lung-substance; unless, between the condensed part and the surface, there exist such quantity of healthy tissue as to mask the morbid sounds by its own natural ones,—or, unless fluid, solid, or gaseous accumulation in the pleura interfere with the conduction of the sounds.

189. In *blowing respiration* both sounds are, as the name indicates, of blowing or whiffing character; and, in its most marked degrees, a sensation as if the air were drawn during inspiration from the observer's ear, or from the surface of the chest, and puffed back during expiration, is distinctly perceived. Both sounds are continuous, *rougher* and *harder*, and especially *more dry* than in altered states of less-advanced type, and more or less *metallic*. The expiratory sound is of much *greater duration* than in the natural state: the inspiratory varies in this respect; both are more intense and of higher *pitch* than natural; and in both, *quickness* of production and progress constitutes, in the most marked forms especially, a characteristic feature.

190. There are two main varieties of blowing respiration: the simple and the hollow.

191. *Simple blowing respiration* is commonly associated by writers with the bronchial variety. This seems an injudicious attempt at simplification; for the two species not only differ in intensity, but in quality (mere bronchial respiration is never metallic), in quickness of evolution, and in pitch;—it seems, too, that the term bronchial should be limited to morbid respiration, simulating that naturally heard in the bronchial tubes.

Simple blowing respiration occurs in two forms,—the *diffused* and the *tubular*. If to the description above given be added the qualification that the whiffing sounds appear to be produced with but moderate intensity, and sometimes at a distance from the ear, over a tolerably extended space, the description of the *diffused* form will be completed. In the *tubular* form, on the contrary, the phenomena appear to occur in a space limited to the immediate neighbourhood of the part examined, and that space to be of tubular shape, cylindrical or flattened. The metallic character is highly developed, to such a degree that the sounds may, without exaggeration, be compared with those produced by blowing sharply through a brass tube: their *dryness*, *rapidity of production* and *intensity* are still greater than in the *diffused* variety. It is in the tubular variety, too, that the sensation of air being drawn from, and puffed back towards, the ear is most distinctly marked.

The tubular form occurs in perfection in but one condition of lung, that of hepatisation; so true is this, that tubular and pneumonic breathing may be used as convertible phrases. But not unfrequently pneumonia runs its course without having produced true tubular breathing,—diffused blowing alone being audible. Now it appears probable, although I am unable to support the notion by reference to post-mortem examinations, that the *diffused* blowing respiration is transmitted from a number of small bronchi, the *tubular* from a few large ones. Were this the case, the change in character from *diffused* to *tubular blowing* would be referable to the compression and obliteration of the smaller bronchi, corresponding to the increase in extent and amount of solidification, and would account for the fact, which may, I think, be frequently observed, that the well-marked *tubular* form signifies a more advanced degree of disease than the *diffused:* great frequency of respiration tends also to give the sharpness of the former to the latter variety. Less perfect tubular breathing is heard in cases of pulmonary abscess, or solid accumulation of moderate size, near the larger bronchi. The diffused form occurs, or may occur, in all conditions of diffused solid consolidation, intra or extra-pulmonary, simple, tuberculous, carcinomatous, fibro-fatty, &c., and in dilatation of the bronchi. There is, besides, a condition under which I have frequently observed the diffused variety of blowing respiration, and where it is likely, unless the examination be conducted carefully, to deceive the observer. In certain cases of tuberculous excavation, auscultation may be performed in two or three places without cavernous respiration with the hollow metallic character being discovered, the diffused variety of blowing respiration being the condition observed: were further investigation not made in such cases, the lung might be considered the seat of simple solidification. By moving the stethoscope carefully over the entire surface, cavernous respiration is usually detected, and one evidence of excavation established. In these cases the cavernous character is evidently masked by the diffused blowing, which is the result of the condensation existing around the excavation. This is one reason why a cavity may escape discovery, unless the examination of the chest be very carefully performed. On the other hand, in certain cases of pneumonia of the anterior apex, of tumor connected with a large bronchus, and of dilated bronchi, true blowing respiration may acquire a hollow character, and hence simulate cavernous breathing.

Lastly, in pleuritic effusion, if there be adhesion or agglutination of the pleura, respiration of the diffused blowing type, often sufficiently marked to suggest the idea of hepatisation, is more or less extensively audible; the presence of condensed lung near the surface sufficiently explains its existence. But it is not alone in these cases of adhesion, that blowing respiration may attend pleuritic effusion; it may be present where no adhesion exists, and the effusion is abundant. It does not, as far as I have known, pass into the tubular variety under these circumstances, is never intense, and in the majority of cases, very slight, especially when compared with the amount of percussion-dulness present; it is also deep-seated, except in the close vicinity of the spine, and generally limited to the middle height of the back—in other words, to the neighbourhood of the main bronchi: in some cases it has all the characters of, and is obviously, transmitted pharyngeal respiration.

192. The theory of bronchial and blowing respiration commonly admitted

regards it as transmitted natural bronchial breathing. Either the sounds of the air-current in the larger bronchi of the solidified lung, or (inasmuch as the respiration-play of that organ is more or less diminished, and therefore little or no flux and reflux of air can occur even in its medium-sized tubes) the sounds of the air-current in the trachea and in the main bronchi of the non-affected lung, are supposed to be conveyed thence through the hepatised tissue with an intensity varying directly as the increase by consolidation in its conducting power. Three objections occur to me as negativing either explanation: the sounds in pneumonic tubular breathing habitually differ in quality and pitch from those of the trachea and larynx; they are commonly much stronger than the latter; while in some rare cases of perfect consolidation, without obstruction of the main bronchus, there is dead silence over the hepatised structure. The conducting power, in regard of breathing-sounds, of hepatised tissue removed from the body, is found by experiment to vary inexplicably.

Skoda* appears to hold that the laryngo-tracheal sounds and those of both main bronchi consonate in the air contained within the bronchi permeating the consolidated parenchyma, and thus produce the phenomenon of bronchial respiration, when intense and of high pitch; whereas weak bronchial respiration of low pitch comes directly without consonance from the lower part of the trachea, the main bronchus, or one of those of second order. It seems doubtful whether this theory can be the true one; for the pitch of metallic pneumonic respiration is irregularly higher than of the laryngo-tracheal, whereas consonating sounds are always either of the same pitch, or of pitches bearing a certain harmonic relation to each other.

The difficulties to be explained are obviously the greater intensity of the thoracic than of the tracheal breathing-sounds, and the irregular differences in their relative pitch. As the same difficulties occur, and appear similarly explicable, in the theory of morbid resonance of the voice, the reader is referred to the discussion on the latter subject [259—274].

193. The essential peculiarity of *hollow* respiration is signified by its name. It is of two kinds, differing only in scale,—cavernous and amphoric, small and large.

194. In the *cavernous* kind the ear receives the impression most distinctly of connection with a hollow space; the *character* of the sounds is *hollow*, whiffing, and moderately metallic; their pitch lower than in tubular breathing. The peculiar *quickness* of production is less marked than in either kind of simple blowing respiration,—there seems to be a delay in the interior of the chest. Cavernous respiration is rarely attended

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* Auscultation, vierte Auflage, pp. 91 and 104.

with any very notable degree of the sensation of air being drawn from the surface and puffed back again. This peculiarity does, however, sometimes exist, and appears to announce the close vicinity of the seat of the phenomenon to the surface.

The veiled puff (*souffle voilé*) is a modified cavernous respiration, in which a "sort of moveable veil interposed between an excavation and the ear" seems to be agitated to and fro: such is Laennec's description of a phenomenon so rare, that I had never, until quite recently, met with it.*

Globularly and largely dilated bronchial tubes, and excavations in the substance of the lung (the less fluid they contain, the more solid their walls, and the freer their communication with the bronchi, the better) give cavernous respiration. Tuberculous excavation is by far its most frequent cause,—cavities produced by gangrene, abscess, cancer, pulmonary apoplexy, and intra-thoracic purulent collections perforating the lung, much rarer ones.

The mechanism of cavernous respiration is probably of at least two kinds. If the influx and efflux of air into and from the hollow space concerned be free, the quality of the sounds is simply explicable by the form of that space. If from rigidity of the thoracic walls, or other physical impediments, the cavity itself can scarcely be supposed the seat of active movement of air, the cavernous quality is probably that of the neighbouring bronchial breathing modified and reinforced within the excavation. Among the physical conditions most favourable to the perfect development of the different varieties of blowing respiration, is emptiness of the space in which they are actually produced, and from which they are transmitted to the ear. This is true of all the varieties; and hence true, whether the seat of the phenomenon be the bronchi, pulmonary excavation, or the pleural cavity communicating by a perforation with the bronchi. If the bronchi or pulmonary cavity contain fluid, the tendency will be to the production of various liquid rhonchi instead; and hence the phenomena of cavernous rhonchus and cavernous respiration are habitually in the inverse ratio of each other, as regards degree and perfection of development. If an excavation be partly filled with fluid and partly with air, the possible conditions are of three kinds: *a.* When the quantity of liquid is small, and the bronchus communicating with the excavation opens above the level of the liquid, pure cavernous respiration will be heard: *b.* When the communication with the bronchus occurs below the surface of the liquid, cavernous rhonchus is heard alone: *c.* When a double communication exists, that is, above and below the surfaces of the fluid, cavernous rhonchus and respiration will be both present. All this has been *observed* by myself and others.

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* Tagg, U.C.H., Females, vol. ix. p. 108.

The cavernous quality is often, confessedly, deficient, where excavations actually exist. Is it ever present, when they are absent? In a well-marked form, I believe, never. Still, there certainly are instances of solidification of tissue surrounding bronchi, unchanged apparently in calibre, in which the tubular quality may be quasi-cavernous: it must be so, in fact; for those bronchi, blocked up at one end, are assimilable to so many elongated cavities. But shall we for this reason reject cavernous respiration as a variety, and blot out the term from our phraseology? No. For auscultation, as an art, is based on the sole teachings of the ear; and the cavernous character is an audible reality. We will simply bear in mind that there are certain rare physical states, rudely imitating excavation of the lung, which furnish, as might be expected, respiration-sounds resembling those of an excavated space.

195. The *special character* of the *amphoric* kind is derived from the attendant sensation of air passing into a large empty cavity having dense walls—such as is perceived on blowing into a water-croft. It is strongly metallic, and sometimes, but rarely, associated in one and the same respiration with metallic tinkling. The amphoric character accompanies both sounds,

but especially the expiratory, though occasionally more markedly the inspiratory; in some instances it only appears on forced breathing; in rare cases it may be heard even at a distance from the chest.* Commonly audible at the middle height of the chest, rare at the upper part, and yet rarer at the base, its physical cause and theory are much the same as of cavernous breathing; the cavity concerned is simply larger. Hence it *may* exist in tuberculous or other excavations in the lung, but they are rarely of sufficient size; broncho-pleural fistula with hydro-pneumo-thorax is its common anatomical cause, whether produced, as is by far most usual, by tuberculous perforation of the lung and pleura, or by perforation of the lung attending the transit of pus either from the pleural sac or from distant parts. The communication with the bronchi must, for perfection of the sign, be above the level of any fluid in the echoing cavity.

Adventitious sounds, produced within the thorax by the act of breathing.

196. The essential difference between the morbid states of respiration hitherto considered, and the sounds now to be described, is, that, in the former, modifications only of a natural sound exist, while in the latter there is actual generation of new phenomena. These adventitious sounds may be produced in (*a*) The air passages, or cavities communicating with these; (*b*) The lung-substance; (*c*) The pleural cavities; (*d*) The mediastina; (*e*) The thoracic parietes; (*f*) The neighbouring organs.

197. (*a*) *Adventitious sounds originating in the air-passages* (*rhonchi or rattles.*)—A rhonchus may be defined as an adventitious sound, audible in inspiration or in expiration, or in both; of dry or moist character; masking the natural murmurs more or less completely; persistent or intermittent; originating in the air-

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* In a case by Louis (Phthisie, 2ème éd. p. 406), at 2¼ inches' distance.

cells, the minute or the larger bronchi, the trachea or larynx, and in excavations of the pulmonary substance; and produced either by the passage of air along bronchi of altered calibre, by air bubbling through fluid contained in the sites mentioned, or by the vibration of semi-solid plastic matter in the tubes, or of prominent folds of their own lining membrane.

198. The audible characters of rhonchi divide them into the genera and species, thus—

- Whistling
  - high-pitched.
  - low-pitched.
- Crepitating.
- Crackling
  - dry.
  - moist.
- Bubbling
  - small
  - large
    - simple.
    - hollow.

199. Whistling rhonchus is either of high or low pitch,—in the former case called *sibilant*, in the latter *sonorous*.

200. *Sibilant* rhonchus is dry-sounding; commonly co-existent with both inspiration and expiration, but especially marked in the former, and occasionally limited to either, it varies much in intensity and duration; recurs irregularly, instead of accompanying every respiratory movement; and is high-pitched,—sometimes so much so, as to be *hissing* in character.

Laennec taught that the sibilant rhonchus of acute bronchitis depended on diminution of the calibre of the bronchi, caused in turn by inflammatory thickening of their lining membrane and subjacent tissue. The natural intermittence of the rhonchus argues strongly against this notion, as the thickening of tissue must be a persistent state; and it is further opposed by the fact that whistling rhonchus may be interrupted and kept off for a variable number of respirations in any given spot, by causing the patient to cough. Coughing, we can readily understand, will alter the position of viscid mucus; but it cannot be supposed to have any direct and immediate influence on the thickness of the mucous membrane, or even on the conditions of a vibrating fold of its substance. The rhonchus, though dry to the ear, clearly depends on the influence of air on fluid, and may be imitated by blowing through saliva between the lips at a certain amount of separation.

When the cause, at least remote cause, of the sibilant rhonchus is of a permanent nature, as diminished calibre from the presence of a tumor, it is said that the rhonchus is itself permanent. But I have, even in cases of this kind, found that the morbid sound will be altogether removed for a while by the act of coughing; and hence believe that, in cases of the sort, the altered form of the bronchus has less to do with the production of the rhonchus, than has the existence of local accumulation of viscid mucus, whether this be a mechanical result of the pressure of the tumor, or the effect of circumscribed supersecretion. If this notion be correct, it is manifest that rhonchi of this class have no claim in respect of their mechanism to the title of dry: the impression made upon the ear alone justifies the application of the term.

201. *Sonorous rhonchus*, varying in special character, is always marked by graveness of tone and dryness; usually co-existing both with inspiration and expiration, but especially marked in the latter, to which it may be limited; varying in intensity from a very slight sound to one loud enough to be audible at some distance from the chest, and to be attended with thoracic fremitus over a surface of variable extent; varying in duration, but having a natural tendency to prolongation; continuously and steadily evolved unless of very short duration, when it is produced in a quick and abrupt manner; occurring interruptedly, or, in rare instances, attending every successive respiration; and sometimes alternating with the sibilant or with bubbling rhonchus.

The special character of the sonorous rhonchus varies; it may be *snoring*, *rubbing*, or *cooing*.

202. The essential cause of whistling rhonchus is the vibration of secreted matters in the tubes—viscid mucus, plastic sub-

stance, &c.: in certain positions it may be produced by the vibration of a thickened fold of the mucous membrane itself: irregular and instantaneous spasm of the smaller tubes frequently induces it, by narrowing abruptly the diameter of the passages through which the air moves: and it may in some instances indirectly proceed from permanent constriction of the tubes, caused either by disease of their coats or by pressure from without.

203. As a general rule, the low-pitched sonorous rhonchus originates in the large, the high-pitched sibilant in the small, the very acute hissing in the minutest, bronchi. But as large tubes may, practically speaking, be made small ones by disease, sibilation may and does occur in bronchi of second and third orders.

204. *Crepitating* (or *crepitant*) rhonchus, when developed in perfection, occurs in puffs more or less prolonged, but rapidly evolved, composed of a variable, sometimes immense, number of sharp crackling sounds, all perfectly similar to each other; conveying the notion of minute size; dry; co-existing exclusively, except in rare cases, with inspiration; and once established, remaining a persistent condition until superseded by other phenomena.

The most accurate comparison which has been made between the crepitant rhonchus and any other species of sound seems to me unquestionably that of Dr. Williams, who likens it to the noise produced by rubbing slowly and firmly between the finger and thumb a lock of one's hair near the ear. In every respect, both as regards the crepiti themselves and the entire act of crepitation, the similarity amounts almost to identity; but it must be remembered that it is to the perfect crepitation of pneumonia, and to this alone, that the comparison is to be understood to refer. Dryness is one of the most marked properties of true crepitation; the sensation is not that of bubbles bursting, but rather of delicate tissue undergoing minutest ruptures with a crackling noise in many points simultaneously. Again, rapidity of evolution is an important property of crepitant rhonchus, and among other characters serves to distinguish it from the small bubbling species: the crepiti appear to be nevertheless successively produced in tissue nearer and nearer to the ear. So, too, persistency is a feature of some importance as distinctive of crepitant rhonchus: other rhonchi are manifestly influenced in the regularity of their production by the occurrence of expectoration, for example; but over true crepitation this appears to exercise no immediate control. The first effect of a fit of coughing, indeed, is to render the rhonchus more distinct and abundant even than before; deep inspiration produces the same effect: under both circumstances parts of the lung, passive in ordinary breathing, are suddenly expanded.

When at its maximum crepitant rhonchus accompanies almost the entire act of inspiration; when first developed, and when about to be superseded by blowing respiration, it appears towards the close of inspiration only. Under all circumstances it is, to say the least, rare to find this rhonchus co-existent in any degree with expiration; the statement that it may generally be heard to a diminished amount with this division of the respiratory act, appears to me to have originated in the confusion which long prevailed between the crepitant rhonchus of pneumonia and the small bubbling rhonchus of capillary bronchitis.

205. Three hypotheses suggest themselves in explanation of the mechanism of this rhonchus. It may be caused by the bursting of minute bubbles in the air-cells and terminal tubes; or by the sudden expansion of the cells, unfolding with crackling noise minute portions of exudation seated between the vesicles in the actual parenchyma; or by the mere abrupt unfolding of the walls of the vesicles themselves. Now, although both the latter theories may be plausibly defended, the first, as originally taught by Laennec, appears to be most probably the true one.*

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* The bubbling theory, however, fails to explain the limitation of the rhonchus to inspiration; while the second of those given above meets this difficulty. For

206. And if this theory be well founded, there is no *à priori* reason, why crepitant rhonchus should not occur in any pulmonary affection, where fluid may accumulate in the vesicles. But as matter of clinical experience the combination of rhonchal characters described is almost peculiar to the early stage of pneumonia; whether this depends on any special qualities of the fluid in that disease, or on the rarity with which the actual air-cells contain fluid in any other affections, is yet open to inquiry. I long indeed, with Laennec, believed fine crepitation distinctive of pneumonia; but unfortunately in rare instances (I have seen two such within the last year) pure œdema of the lung does give crepitant rhonchus in no point distinguishable from that of pneumonia. I have not myself met with true crepitation in pulmonary apoplexy, or in the common hæmoptysis of phthisis.

207. *Crackling* rhonchus is *dry* or *moist* in character.

208. The *dry crackling rhonchus* is composed of a succession of minute, dry, short, sharp, crackling sounds, few in number, rarely exceeding three or four in a respiration; coexisting exclusively, or almost exclusively, with inspiration, though in very rare cases most obvious in expiration, especially when it has existed for some time; permanent, in the great majority of cases, after its characters have once been perfectly developed, until it ceases altogether to be produced, in consequence of its

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though the rapid and abrupt unfolding of the glutinous mass be productive of crackling noise, it is very unlikely that the comparatively slow and equable restoration of the tissue to its previous collapsed state would be thus productive, —indeed the presumed physical cause of crepitus has ceased to exist. On the other hand, there is no reason why on the bubbling theory crepitation should not as regularly exist, though not with the same loudness, in expiration as in inspiration. The air is presumed in inspiration to have passed through a certain fluid; if so, it must repass through it during expiration, and assuredly with a noise similar in kind, though less in degree: when rhonchi are manifestly produced by the passage of air through liquid, as in the bronchial tubes or in excavations, they attend both inspiration and expiration. Further, the nature and mechanism of pleural, mediastinal, and intra-parenchymatous pseudo-rhonchi oppose the first, or bubbling, theory.

passing into the moist variety; and usually conveying the impression to the ear of being evolved at a distance from the surface.

In stating that the dry crackling rhonchus, once *perfectly* developed, remains commonly a persistent condition until the transition into the moist form is established, I wish to lay particular stress on the circumstance of its being so developed. While yet producible only by forced respiration, and appearing only with an occasional inspiration, it is liable to disappear for a day or two, and then recur; but when once it has acquired sufficient perfection and stability to maintain itself steadily through a number of ordinary respirations, it apparently constitutes a persistent state.

This rhonchus, though its mechanism be unexplained, is of considerable diagnostic importance. It is of tolerably frequent appearance on the eve of the softening process in tubercles, and *may* hence be discovered wherever that product exists in the first stage. In the great majority of cases it is found in the infra-clavicular, or supra-scapular regions,—and most commonly in the former. Its natural course is to pass into moist crackling; and, according to M. Fournet, in cases of acute phthisis, the transformation occurs in the majority of instances in from eight to twenty days: in from twenty days to two months and a half, or three months, in the chronic form of the disease. These statements respecting time must be received with caution, however, and are subject, as admitted indeed by this writer himself, to very distinct exceptions.

209. *Moist crackling.*—A rhonchus composed of a series of clicking sounds,—few in number,—of moderate size,—occurring during both respiratory movements, but with greater regularity and distinctness of character in inspiration, and eventually passing into, or rather superseded by, rhonchi of the bubbling class.

Although this rhonchus may be very satisfactorily connected clinically with incipient softening of tubercle, its mechanism is almost as obscure as that of the dry crackling. It appears rather to be produced in direct connection with tuberculous matter, which has commenced to undergo softening, than to constitute a mere form of bronchial rhonchus, having its seat in the bronchi, and produced by bubbling of air through mucus. The clicking character it possesses, combined with the special nature of the anatomical conditions which it is known to attend, points to a peculiarity in its mechanism. Possibly it originates in the interior of softening tubercles which have just commenced to communicate with the minute bronchi. If so, its "conversion" into bubbling rhonchus would in reality be nothing more than its being superseded by that state.

210. *Bubbling* rhonchus is *simple* or *hollow* in character. The simple kind varies in the size of its component bubbles, which are *small* or *large*,—a subdivision of clinical significance.

211. In *small-sized bubbling* (sub-crepitant) rhonchus, the component sounds have a distinctly bubbling character ; they are of moderate size ; humid ; scarcely ever occur in puffs ; are evolved with variable quickness, but rarely with much rapidity ; are few in number and dissimilar to each other ; occur with more or less irregularity ; and attend both respiratory movements. The bubbles vary in size and liquidness.

Produced by bubbling of air through liquid of variable consistence in minute bronchial tubes, this rhonchus, if occurring at both bases posteriorly, indicates idiopathic capillary bronchitis,—if at either apex, tuberculous bronchitis,—if at one base posteriorly, it most commonly depends either on pneumonia in a state of resolution, or on bronchitis attending tubercle of the *upper* lobe, though occasionally connected with emphysema in excess on the same side. With very liquid bubbles it occurs in various parts of the chest from pulmonary apoplexy,—at both bases posteriorly from idiopathic or post-pneumonic œdema. Auscultators are indebted to M. Louis for establishing its frequency, at both bases, as a sign of capillary bronchitis.

212. In the *large-sized bubbling* rhonchus (so-called *mucous*,

submucous, &c.), the bubbles are of variable size, but all materially larger than in the last described species, variable in number, and unequal in size,—distinctly liquid,—irregular in recurrence, —modified by the acts of coughing and of expectorating, and co-existing with both respiratory movements. The size of the bubbles localises the rhonchus in the larger or smaller bronchi.

Caused by bubbling of air through liquid (mucus, serosity, blood, pus) contained in tubes of moderate or considerable calibre, and most commonly audible towards the central parts of the lungs, this rhonchus attends the secretion-stage of bronchitis, bronchorrhœa, dilatation of the bronchi, bronchial hæmorrhage, hæmoptysis, evacuation of pus from the pleura or elsewhere through the bronchi, and occasionally, the suppurative stage of pneumonia.

213. *Hollow bubbling rhonchus* (cavernous or gurgling), consists of a limited number of bubbles of large size, distinctly liquid, occasionally disappearing for a time, having a peculiar ringing hollow metallic character, and co-existing commonly with inspiration and expiration,—in some cases with either alone,—and associated or not with cavernous respiration. The size of the bubbles varies; whence the rhonchus has been called *amphoric*, *cavernous*, and *cavernulous*. In the last and smallest there is distinctly a clear metallic character, but the sensation of hollowness is not perceived, or at least imperfectly. Cavernous rhonchus may be sometimes heard at a distance from the patient's chest.

The causes of temporary cessation of cavernous rhonchus are —1. Complete evacuation of the liquid contents of the space in which it is produced; under these circumstances it is replaced by cavernous respiration; 2. Diminution of the contents to such extent as to bring the level of these below the bronchial opening or openings into the cavity; 3. Absence of air in the cavity, the entire space being filled with fluid; 4. Obstruction, by inspissated muco-pus or otherwise, of the bronchi communicating with the cavity; 5. Pressure of the lung by pleuritic effusion,*—a rare cause, seeing that the surface of the excavated parts is commonly agglutinated to the costal pleura.

Whenever bubbles burst in a hollow space within the chest, the resulting clicks resound and are echoed by the walls of that space,—the harder and smoother the walls, the more perfect the echo; the softer and more rugged, the less perfect. So true is this, that excavations of small size, and surrounded with even tolerably healthy parenchyma, furnish no real hollow rhonchus,—the bubbles form, but their clicks are not echoed. The common cause of bubbling is, of course, the passage of air in and out of the excavation; and, according to the relative position of the contained fluid and the communicating bronchi, will the period of the acts of respiration, at which the rhonchus is best heard, vary. The bubbles may sometimes probably burst at the bronchial orifices, and then echo within the cavity. Besides this, a species of gurgling, resembling cavernous rhonchus somewhat, and becoming metallic in quality, if the mouth be held open, may be produced in large cavities from the agitation of their contents by the impulse of the heart.† I have only heard this in the left lung, but Dr. Stokes has observed it in both, and even in the posterior portion of the right lung, producing a "tick loud enough to reckon the pulse by." Under all circumstances it is rare,—even when the heart and cavities are closely contiguous.

Excavations from tubercle are, of course, the most common anatomical state present; but those of abscess, sphacelus, softened cancer, pulmonary apoplexy, and perforating empyema, &c., as likewise dilatations of the bronchi, may be the seat of the phenomenon.

214. *Dry crepitant rhonchus with large bubbles*, "observed only during inspiration, conveys the impression as of air entering and distending lungs which had been dried, and of which the cells had been very unequally dilated, and entirely resembles

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* Stafford, Consumption Hospital, Chelsea, in "Lancet," July, 1844.

† Jamieson, U. C. H., Males.

the sound produced by blowing into a dried bladder." Such is Laennec's description of a "rhonchus" which he believed peculiar to emphysema, pulmonary and interlobular; the phenomenon is excessively rare, and cannot be correctly spoken of as a rhonchus,—it is rather a form of respiration, distinguished by dryness and crispness, and is totally wanting in bubbling character. It mainly indicates the existence of dry distended pouches under the pulmonary pleura,—the most advanced condition of pulmonary emphysema. In the few instances in which I have heard the sound, it was transitory.*

215. Under the title of consonating Skoda describes a rhonchus, clear, high-pitched and unequal-bubbled, accompanied with resonance, which is, however, neither metallic nor amphoric. It is found along with bronchial breathing and bronchophony, and generally signifies pneumonia or tuberculous infiltration. This description would answer to bubbling rhonchus, produced in tubes surrounded by solid tissue. That a rhonchus, produced in one part of the air-passages, can be reinforced by consonance, to an amount clinically appreciable, in another and distant one, remains, I think, to be proved. Nor do I find that Skoda gives any satisfactory rules by which the alleged dependence of a rhonchus on consonance can be established during life.

216. (*b*) *Adventitious sounds originating in the lung-substance* (*pulmonary pseudo-rhonchi*). If individuals, whose lungs are healthy, or diseased only at the apices, and whose breathing is habitually calm, are made suddenly to respire deeply, a peculiar, fine, dry crepitation, accompanying inspiration only, may often be detected at the bases posteriorly. But after two or three, or at most five or six, acts of respiration, it totally disappears. This pseudo-rhonchal sound seems to depend on the sudden and forced unfolding of air-cells, which are unaffected by the calm breathing habitual to the individual; and its only importance arises from the possibility of confounding it with crepitant rhonchus.

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* Hayes, U. C. H., Females, vol. v. p. 24.

Here we have a minor degree of the phenomenon observable in the same regions, when any considerable portion of the base of the lung is under the influence of persistent pressure from tumors or enlarged abdominal organs.—I mean the pulmonary compression pseudo-rhonchus,* which consists of a series of fine, very dry crepiti, evolved at a peculiarly slow and drawling pace, variable in number, but generally very numerous, and commencing towards the close of inspiration, or in some cases apparently when this movement has almost ceased.

217. This sound of unfolding lung varies in quality, if the lung be diseased. Thus, in a case of arrested phthisis, at the apex of the lung, where, nine months before, softening-signs were most manifest, I have recently found dulness under percussion, feeble bronchial breathing, and a deep-seated creaking sound accompanying inspiration: in all probability the unfolding of induration-matter in the lung is the cause of this sound. I have repeatedly observed this sign, without having traced the progress of its development so well as in this instance.

218. (*c*) *Adventitious sounds in the pleuræ.*—Daily experience proves that the collision of the opposite laminæ of each pleura, during inspiration and expiration, is not in the healthy state productive of appreciable sound. Experiments upon the lower animals might, were this necessary, be referred to in confirmation of the fact. This noiselessness of movement of the pleural surfaces upon each other depends upon their perfect smoothness and slight humidity; when these conditions become changed by disease, their gliding motion is attended by different modifications of sound, varying with the nature and amount of the existing anatomical change. While these different sounds all agree in being produced by friction, some of them, also, convey this sensation to the observer, and are, therefore, commonly designated as *pleural friction-sounds.*

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* Clinical Lectures, loc. cit. p. 525, in case of enlarged liver; also in case of enlarged spleen, . . . Dujardin, U. C. H., Males, vol. v. p. 195.

219. *Pleural friction-sound* consists either of a single, or, more commonly, of a series of abrupt jerking sounds, few in number, and manifestly superficial in seat: it is audible over a variable, but usually limited, extent of surface; persistent or intermittent; of variable, but commonly more or less considerable duration; ranging, in point of intensity, from a scarcely audible noise to one of extreme loudness; attended with a sensation of dryness: almost invariably heard in inspiration, and habitually, but by no means always, more intensely developed with that movement; most frequently accompanying both inspiration and expiration, seldom expiration alone;* produced with ordinary respiration, or developed only after coughing, or by deep inspiration; in strongly-marked cases attended with fremitus palpable to the hand, and perceptible to the patient, and sometimes increased by pressure with the stethoscope. Calm, may give more friction than forced, respiration.†

220. The modifications of intensity and special character of friction-sound justify the establishment of four varieties: *grazing; rubbing; grating; creaking;* peculiarity of rhythm founds another variety, the *rumbling.*

221. *The grazing variety*, the most delicate form of friction, is usually a single sound; audible over a very limited extent of surface; occurring with an occasional respiration only; remarkable for mobility; more rapidly evolved, and of less duration than the other varieties; dry and limited strictly to inspiration. Varying from hour to hour, it may be, in precise site, this variety is almost peculiar to the dry period of pleurisy, occurring rarely at the absorption-period, and is mostly met with in the phthisical form of that inflammation. In primary idiopathic pleuritis it is very rarely to be detected, because the period of

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* Clansey, U. C. H., Males, vol. viii. p. 73. Total limitation to expiration is of temporary duration. Friction sound may also be most prolonged, and consist of the greatest number of jerks in expiration, *e. g.*, Humbert, U. C. H., Males, vol. ix. p. 14; Bassett, U. C. H., Females, vol. vii. p. 226.

† Bassett, U. C. H., Females, vol. vii. p. 229.

its existence has usually passed away before the chest is submitted to examination. In cases of intercurrent pleurisy, where the patient has been under treatment for the primary disease, a better opportunity is afforded of establishing its existence. Another source of difficulty in its detection, even in tuberculous cases, is the shortness of its duration: a single day suffices for its production, development, and termination; and this series of changes may, as I have, though rarely, observed, be accomplished several times successively in the course of a few days.

The ordinary seats of grazing friction are the infra-mammary, infra-axillary, and infra-scapular regions; I have not yet satisfied myself of its existence, either in the supra-spinata, supra-clavicular, or infra-clavicular regions: probably it is masked in these vicinities by the louder morbid sounds usually present. The sign is not only of clinical but of pathological importance, because it has helped to connect the fugitive chest-pains of phthisis with local and evanescent pleurisy.

222. *The rubbing variety*, a more advanced type of friction, consists of a series of jerking sounds, rarely exceeding three or four in number; is audible over a tolerable extent of surface, provided the necessary condition of motion of the lung exist; of rather considerable duration, slowly evolved, attends both inspiration and expiration, and is more frequently than other varieties attended with friction-fremitus. It occurs in acute pleurisy, at the periods of plastic exudation, and of absorption, being more frequently detected at the latter. The mechanism of friction-sound causes a difficulty in conceiving its production, where great liquid effusion is present in the pleura; and ordinary experience is in accordance with *à priori* considerations—the two conditions are not observed to coexist. Dr. Stokes was, however, the first to mention a case in which, though great and universal dulness of the side existed, friction phenomena were audible, and even perceptible to the patient in the postero-inferior and lateral portions of the chest: "they may then," he infers, "coexist with extensive liquid effusion." There is one

ource of fallacy in such cases; supposing them to have reached he period of absorption, the fluid might have been nearly emoved, and yet the condensation of the superficial strata of he lung, combined with an accumulation of plastic exudation-natter, been sufficient to produce extensive and marked dulness. Now, under such circumstances, the production of friction-henomena would have been inevitable. However, the retention f some portion of the lung's surface in tolerably close proximity o the costal pleura, by means of adhesions, also renders the roduction of friction-sound possible, although a considerable uantity of fluid be present in the pleura. M. Fournet states hat he once ascertained the coexistence of these three condiions; and I presume that three examples I have myself observed f coexistent friction-sound, and general effusion-signs, were hus explicable. Effusion-signs may be evident in the back, nd friction-signs in front; this is common.

Laennec described friction-sound as an attendant upon, and ne of the most important diagnostic signs of, interlobular mphysema; while he made no mention of its existence in leurisy. The experience of his followers has reversed the onnection which Laennec sought to establish: it is now elieved that emphysema of any anatomical form is as incapable f producing friction-signs, as pleurisy is indubitably their ommon cause. Andral, Louis, Stokes, Fournet, and other riters, agree in denying, either directly or by inference, that he collision of subpleural vesicles, or of pulmonary septa endered prominent by infiltration of air, against the opposite leura, is an observed cause of friction-sound. Such was the pinion which I held upon the point also; and I still believe hat we want the anatomical proof of the phenomenon being hus generated. But from some cases I have met with of very dvanced emphysema, manifestly attended with a low degree of ubbing-sound at the postero-inferior part of the chest, where ubpleural vesicles are very commonly developed, and presenting either signs nor symptoms indicative of pleurisy, I am induced to think that Laennec's belief respecting the occurrence of friction-signs in some forms of emphysema was not erroneous.* Upon referring to certain cases in my possession of individuals dying with extensive infiltration of air under the pleura, I find the existence of friction-sound during life noted, and certainly not a syllable respecting exudation-matter in the pleura among the details of the post-mortem examination. I can scarcely think that so obvious an appearance as this would have been forgotten, had it existed. These cases, too, are perhaps deserving attention, because collected at a period when I had a preconceived notion against the possibility of friction-sounds being evolved in the manner now referred to. It is true, Dr. Stokes maintains their evolution under the circumstances to be physically impossible. "It is only," he observes, "when the surfaces are rendered dry by an arrest of secretion, or roughened by the effusion of lymph, that their motions produce sounds perceptible to the ear." But this remark, which is perfectly just, if applied to these surfaces when holding to each other their natural relation of simple approximation without mutual pressure, begs the question at issue when applied to pleural laminæ, one of which presents elevations on its surface necessarily productive of some slight pressure against the other.

223. *The grating variety* conveys the sensation indicated by its name; and, except in respect of its greater sharpness, is characterised as the last variety. It may occur at the period of absorption, with or without retraction of the chest, and occasionally that of exudation. It sometimes indicates that the exudation-matter is laid down in the granular form; and may be produced by sub-pleural miliary tubercles, causing superficial prominences.

224. *The creaking variety* is suggestive of the noise produced by the creaking of new leather of moderate hardness; in other respects it resembles the rubbing variety, with which it has a

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* I have (Hayes, U. C. H., Females, vol. v. p. 25,) distinctly observed them in the mammary region.

[illegible] It commonly indicates dryness, roughness, [illegible] on the part of the [illegible], and hardly ever, except in rare [illegible] into the creaking type. The [illegible], that sometimes [illegible] furnishes in rare instances, either by its own [illegible] or by [illegible] with the [illegible] a [illegible] of [illegible], more frequently [illegible] in the [illegible] form.

225. Instead of being composed of a series of distinct interrupted jerks, friction-sound may be prolonged without cessation from the beginning of inspiration to the end of expiration; but [illegible] rarely, evidently allied to pleural pseudo-membrane, the [illegible] of rubbing may be [illegible].*

226. The duration of each [illegible] of friction-sound, [illegible] of which occur in each act of respiration, depends on the extent of surface in a state to furnish the sound, the [illegible] of motion, [illegible] of the lung. The length of [illegible], [illegible] a [illegible] of [illegible], especially during the [illegible] period, [illegible] greatly.

Although the audible characters of friction-sound are, as [illegible] above, somewhat influenced by, they do not bear any [illegible] relationship to, the physical state of the [illegible] generating that sound. We cannot [illegible] from the [illegible] of friction-sound the state of the pleural [illegible],—[illegible] doubtful, that we can affirm, on the evidence of [illegible]-sound, whether this is caused by exudation or by mere [illegible] and [illegible] of surface,—as will be more fully shown in the history of pleurisy.

227. The motion of respiration is, of course, the ordinary [illegible] cause of the rubbing of surfaces required for the [illegible] of pleural friction-sound; but, as first noticed by [illegible] Stokes, the heart's impulse may, under [illegible] circumstances, produce it. The rhythm of ordinary pleural friction [illegible] with the movements of respiration; that of the variety

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* Case of [illegible] Clin. Lectures [illegible] p. [illegible].

generated by cardiac impulse, agrees with the heart's movements; hence a difficulty, elsewhere considered [473], in determining on the pericardial or really pleural origin of such friction.

228. *Pleural Pseudo-rhonchi.*—In the winter of 1842, I made the following observation:—In a male adult presenting the most evident signs, both in front and behind, of a cavity at the left apex, an extremely abundant medium-sized rhonchus occurring almost in puffs, and having the liquid bubbling character in a most marked manner, was day after day, during the week previous to death, detected in the entire height of the left side posteriorly. The rhonchus was, however, distinctly more abundant and more liquid, as noted in writing during life, in the upper scapular and upper part of the lower scapular regions, than elsewhere. As the patient was anasarcous to a high degree, the urine albuminous, and as he constantly lay on the left side, the explanation of the rhonchus naturally suggesting itself was, that it depended on œdema of the pulmonary tissue generally, but most marked at the apex, and there of course affecting tissues lying between the cavity and the surface of the lung. At the post-mortem examination, however, I found this explanation was inadmissible; for the thin lamella of tissue between the cavity and the surface was as hard as cartilage, and contained not a particle of serosity; nor was the organ in any part distinctly infiltrated with fluid, being, on the contrary, particularly dry from its excessive induration. But all along the posterior surface of the pulmonary pleura there appeared, in addition to ordinary dense pseudo-membrane, a quantity of fine adventitious cellular tissue, abundantly infiltrated with liquid. Masses of some size were formed from place to place by the accumulation of fluid in the meshes of this cellular tissue, and it was observed by those present, who had not seen the patient during life, that they were much larger than elsewhere at the apex. There was no air either in the cavity of the pleura, or intermixed with the serosity. Now, although it was possible to suggest another explanation, it seemed most reasonable to

suppose, under the circumstances, that the rhonchal sound was actually produced in the masses of infiltrated tissue referred to, and therefore outside the lung, and independently of air.

Subsequent experience has amply proved the correctness of this explanation, and shown that moist sounds, rhonchoid in properties, are producible whenever adventitious tissue within the pleura is infiltrated with serosity, and the movements of the chest continue free. The sounds occur in two forms, *squashy* and *crackling*. The character of the first is represented by its name, and, coupled with the sensation of extremely superficial site, suffices for its diagnosis.* The crackling form, in itself indistinguishable from some conditions of subcrepitant rhonchus, may be diagnosticated by the coexistence of friction-sounds, constant or occasional, and by its being unaffected by coughing. Mere moisture in plastic matter within the pleura seems enough to give a rhonchoid character to friction-sound.

229. (*d*) *Adventitious sounds in the mediastina.*—Crepitation, inspiratory and expiratory, of variable degrees of dryness, abundance, and size, audible in forced respiration only, or in calm breathing, constant or intermittent, disappearing after a few chest-expansions, or continuing through a long examination, is sometimes to be discovered over the sternum, generally or partially, while it is completely wanting over the contiguous portions of lung. No symptoms of any kind necessarily attend this state.†

How is it produced? When the cellular tissue is infiltrated with serosity and air, the production of such sound through the movements of the chest is quite intelligible. I observed the fact in a remarkable case, where air was infiltrated into the mediastina, through perforation of the œsophagus and pericardium, effected in the attempt to swallow a knife.‡ But whether the presence of air be absolutely requisite, I do not know as matter of experience. This pseudo-rhonchus derives its clinical interest from the likelihood of its being mistaken (as it has actually been) for the crepitant rhonchus of marginal pneumonia.

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* The precise spot where infiltrated false membrane exists may be pointed out during life by this sound: e. g. cases of Griffiths, U. C. H., Males, vol. ii., p. 183; and Donovan, U. C. H., Males, vol. ii. p. 202.

† Scott, U. C. H., Males, July 23, 1850.

‡ Ramo Samee, Univ. Coll. Mus., No. 3859.

230. (*e*) *Adventitious sounds in the thoracic parietes.*—Various sounds generated in the framework of the thorax and its integuments by the breathing movements, aided or not by pressure of the stethoscope, derive interest from the chance of their being confounded with intra-thoracic sounds,—a chance not unfrequently realised. There is scarcely a form of pulmonary morbid sound that may not be thus simulated.

231. Pseudo-crepitation will occur, if the stethoscope be accidentally placed over hair on the surface,—in a less deceptive form, if the subcutaneous cellular tissue be emphysematous. The crepitus of a fractured rib has been mistaken for the rhonchus of pneumonia.

231*. Creaking sounds are sometimes heard about the cartilages, probably from interstitial dryness, as they are by far most common in aged persons; I have known these mistaken for creaking pleural friction-sound.

232. A moist rhonchoid sound, perfectly resembling fine bubbling, is often audible, when the integuments are infiltrated with serosity. This anasarcous sound may disappear totally, when the subjacent fluid has been completely pressed aside by the end of the stethoscope.

233. Pseudo-friction sound is also, though less frequently, producible by anasarca of the chest-walls. The imitation may be distinguished from the reality by its being limited to œdematous spots, by its existing in the abdominal as well as the thoracic walls, by its not being perfectly synchronous with the respiration-movements, and by its disappearing after steady pressure with the stethoscope.*

A sound, imitative of intra-thoracic friction, may be produced by the movements of the scapula in breathing,—probably when the cellular tissue between that bone and the chest-wall is unusually dry. When occurring in the upper fossa, it simulates somewhat the cogged-wheel rhythm [186]. At either fossa it is removable by a few brisk movements of the arm.

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* Peculiarities all of them observed in the case of J. Morris, U. C. H., Males, vol. vii. p. 159.

234. The muscular actions going on in the chest-wall give rise in some persons to a peculiar buzzing rumbling sound; its amount does not appear to be directly as the muscularity of the individual. It is increased by efforts of all kinds,—as for instance, that of maintaining an uncomfortable posture; it is continuous, not rhythmical with respiration; and rather increases, than diminishes, in intensity when the breath is held. It may in some spots be removed by putting the muscles in a state of relaxation; but in the infra-axillary regions, where it is sometimes highly marked, cannot be thus arrested.

235. According to the site of various of the sounds now passed in review, they may, without due care, be taken for evidences of pleurisy, pericarditis, pneumonia, œdema of the lung or phthisis.

236. (*f*) *Adventitious sounds produced in neighbouring organs.*—Very little care will prevent the rhonchoid noise, caused by swallowing saliva, and by intestinal borborygmi from being mistaken for intra-pulmonary sounds. In cases of great gaseous distension of the stomach, the bursting of bubbles within that organ may simulate amphoric rhonchus with echo and metallic tinkling: the percussion-sound will at the same time be extensively amphoric, but not tympanitic, in the left inferior regions; but there will be no amphoric breathing, and the act of swallowing fluid will immediately increase the rhonchoid sounds.

§ II.—The Resonance of the Voice.

I.—IN HEALTH.

237. The particular rules for performing auscultation of the voice are not numerous. The most essential point is, that

its pitch, intensity, and tone be the same, while different points of the chest are examined. Now this uniformity can only be insured by causing the patient to repeat monotonously the same word or syllable: even slight inflection of tone may modify the resonance; and variation in the class of consonants pronounced will have even more certainly the same effect.

238. Generally speaking, the patient's utterance should be loud, while the vocal resonance is examined; but in the auscultation of the voice over caverns, the most decisive information is sometimes obtained from the whisper, beyond which the patient is, in the advanced stages of tuberculous disease, frequently unable to raise his tone.

239. The stethoscope should be laid firmly upon the surface, and the ear similarly applied to the instrument, but without any degree of forcible pressure; if either be too lightly applied, a tremulous bleating character may be given to the resonance; if too forcibly, the distinctness of this is diminished. It is scarcely necessary to add, that the instrument should be used in precisely the same manner and in precisely corresponding spots upon both sides of the thorax. The condition known as exaggerated resonance is perhaps more accurately appreciable by means of immediate than mediate auscultation; all other unnatural states of vocal resonance are more satisfactorily and distinctly ascertained with the stethoscope.

In examining certain regions, great care must be used both in the manner of applying the instrument and in drawing inferences from the results obtained; the chief of these regions are the post-clavicular, the upper sternal, and the inter-scapular. The vicinity of the trachea or large bronchi is, without such care, liable to mislead the observer, on account of the naturally greater resonance to which that vicinity gives rise. When the post-clavicular space is examined, especially its inner part, the stethoscope should be held as nearly as possible parallel to the trachea.

340. If the stethoscope be applied over the larynx or trachea

of a healthy individual while speaking, the voice is transmitted, imperfectly articulated, through the instrument to the ear, with a degree of force, loudness, and concentration so great, that the experiment may be productive of actual pain to the observer, especially if the voice examined be grave and powerful. The same transmission of the voice occurs at the lateral parts of the neck, and even over the spinous processes of the vertebræ behind, but with less intensity than on the middle line in front. The resonance of the voice heard in these situations is called *natural laryngophony* and *tracheophony*. At the upper part of the sternum, on the middle line, the vocal resonance is considerably weaker; towards the edges of the same part of that bone a still further diminution is perceptible, and the resonance is here reduced to the state called *natural bronchophony*. The sound is now more diffused, its articulation very imperfect, its quality like that of the voice heard through a speaking-trumpet; it also appears to be, as it actually is, produced at a greater distance, and no longer seems to strike directly against the ear. This species of resonance is also observable posteriorly on the middle line over the division of the trachea, and on either side of that division between the spines of the scapulæ. Over the parietes of the chest, with the exception of the parts just referred to, the natural resonance of the voice amounts to no more than an obscure buzzing, unattended with any approach to articulation, and appearing to reach the ear from, rather than to originate at, a certain depth within the chest, and from an indeterminate extent of surface: in many persons even this obscure buzzing is wholly deficient.

241. The intensity and quality of this natural resonance are modified by certain circumstances altogether independent of disease. Thus, the natural resonance is *cæteris paribus* marked in proportion to the graveness of the voice. This statement is only true of intensity, however; there is no greater tendency to concentration or articulation of the sound when the voice is grave than when it is shrill: Secondly, vocal resonance is, as a

corollary from the last proposition, more marked in males and in adults than in females and in children; it is also more marked in aged persons than in adults, probably on account of the thickening and hardening of the bronchi in old age: Thirdly, the quality of the resonance varies with the quality of the speaking voice; thus in people of advanced age it is very commonly tremulous and bleating: Fourthly, the resonance is more strongly developed, the larger the chest, and the less loaded its walls with fat and muscle: Fifthly, it is stronger in front than behind, with the exception of the interscapular region; and at the upper than the lower parts of the thorax: Sixthly, as first stated by Dr. Stokes, its intensity is greater on the right side generally than the left,—a fact chiefly significant, as noticed by Sir James Clark and M. Louis, under the clavicles and in the interscapular regions; there is no resonance over the superficial cardiac region, nor over the hepatic surface below the sixth rib: Seventhly, the intensity of vocal resonance, as of the respiratory sounds, varies much in persons apparently presenting the same physical conditions for its development;—hence the danger of drawing any inference directly from the state of vocal resonance in a given portion of the chest; it is only by the application of the principle of comparison of the two sides that any safe result can be obtained, due allowance also being made for their natural differences.

241*. The want of uniformity between vocal resonance and vocal fremitus has already been spoken of [43]. 45

II.—IN DISEASE.

242. The signs derived from modified vocal resonance are uncertain in character and obscure in theory, and, though occasionally not devoid of clinical signification, hold, as a rule, a very low place among physical aids to diagnosis.

243. The natural vocal resonance may be diminished or increased in intensity, without or with alteration in quality. Its perversions may be arranged thus:

Diminished intensity	Weak	
	Suppressed.	
Increased intensity	Exaggerated	
	Bronchophony	Simple
		Pectoriloquous—amphoric.
		Ægophonic.

244. Diminution of resonance varies between weakness and suppression. *Weak resonance*, as its name implies, signifies a state in which the vocal resonance is distinctly less marked than natural; commonly spoken of as rare, it is really of frequent occurrence. Thus in the dilatation-period of liquid and aeriform effusion into the pleura, in cases of obstructed main bronchus, even in solidification of the lung, and when excavations have formed, the corresponding resonance may be temporarily weaker than natural.

245. *Suppressed resonance* means that rare condition in which all transmission of the voice to parts of the surface, where naturally perceptible, has ceased; no audible sound being conveyed there. Impairment of vocal resonance exists in emphysema; and in pneumothorax, simple or fistulous, actual suppression may occur. But neither of these conditions is constant: in emphysema the resonance may be extra-powerful. Simple pneumo-thorax is so rare, that we have little opportunity of substantiating its signs, and special peculiarities occur in the fistulous variety. During the dilatation-period of pleuritic effusion the resonance may be weakened even to suppression.

246. Increased resonance presents itself in the forms of *exaggerated resonance* and *bronchophony*, which are little more than forms of each other. The intensity of sound in the former undergoes simple increase; in the latter there is, besides, just as in the natural resonance over the main bronchi, a greater concentration of sound: the former may be considered a *diffused* bronchophony; the latter a *concentrated* resonance of exaggerated force. In bronchophony the sound is also clearer, and more distinct; unattended with distinct articulation; and occa-

sionally producing a quivering sensation in the ear of the observer. Both are usually constant phenomena, so long as they exist; that is, produced every time the patient speaks. The area of bronchophony may, or may not be, sharply defined; in other words, the transition from the most distinct bronchophony to the natural resonance may be sudden; or, as is more common, a gradual diminution of the phenomenon is traceable. In quality it may be metallic, sniffling, or partake, chiefly in aged persons, of the tremulous and bleating tone especially characterising ægophony. As in the case of natural resonance, there is no uniform ratio between the strength of bronchophony and of tactile fremitus.

247. The physical conditions in which simple bronchophony is clinically observable are:—1. Increased density of the pulmonary tissue either surrounding pervious bronchi, or forming a medium of communication between pervious bronchi and the spot of the thoracic surface examined, whether that increase of density be caused by solid, semi-solid, or even liquid infiltration of the parenchyma. 2. Increased density of texture, produced by extraneous pressure. 3. Presence of any solid extra-pulmonary formation in such a situation as to form a connecting link between the surface examined and a bronchus of some width. 4. Increased width and hypertrophy of the substance of the bronchial tubes. 5. Diminished density of the lung, as in the rarefaction of vesicular emphysema. 6. Excavation of the lung. The diseases referable to the first head are the common causes of bronchophony, but any hypothesis in explanation of the phenomenon must also apply to the other and comparatively rare causes.

Thus in its maximum degree, and marked by the metallic and sniffling quality, bronchophony coexists with tubular blowing respiration, in the parts corresponding to the hepatised lung. In the non-metallic form it is heard in tuberculous and plastic consolidations, in pulmonary apoplexy, slightly in pulmonary œdema. It exists in dilatation of the bronchi; but the enlarged caliber of the tubes is not habitually the sole condition of its presence; coexisting thickening, and hardening of their walls, and condensation, attended with chronic induration of more or less pulmonary substance around, contribute materially to its production: still, increased width may be the sole appreciable cause of the sign. Bronchophony cannot be regarded as an ordinary sign of pleurisy with effusion. At the stage of effusion with general dilatation of the chest, when a broad mass of fluid intervenes between the lung and parietes, all vocal resonance has ceased; but when the effusion is moderate, this phenomenon may be detected under certain circumstances. Thus, in the immediate vicinity of the larger bronchi between the scapulæ, bronchophonic resonance is generally to be found. So, too, if there be induration of pulmonary substance, superadded to the mere condensation from pressure, bronchophony may be sometimes very manifest posteriorly and laterally; for example, where slight hepatisation coexists with effusion. Bronchophony may often be heard in the upper front regions, when the effusion is sufficient to condense a considerable portion of the lung inferiorly. Again, it may be detected, as affirmed by M. Reynaud, at a part of the surface which had just given ægophonic resonance, by causing the patient to alter his position in such manner as to displace the pleural fluid from the spot under examination. It is common, also, at the period of absorption and retraction of the chest-walls; and from the existence of partial pleural agglutinations or adhesions, bronchophony is sometimes locally audible throughout the entire course of effusion. Compressed texture, whatever be the cause of compression, may give bronchophonic resonance, if of any notable bulk. The connection of bronchophony with excavations will be by-and-by considered.

248. There are two conditions of vocal resonance, described by Laennec under the names of pectoriloquy and ægophony, the nature and significance of which are yet *sub judice*.

249. The essential character of pectoriloquy is, according to

its discoverer, complete transmission of the voice through the stethoscope,—that is, a sensation as if the words uttered passed directly into the ear of the observer from the spot beneath. This condition of resonance he believed to be peculiar to excavations in the substance of the lung, and, besides, an invariable attendant on them, except when extraneous circumstances interfered with its production or propagation. As, however, he repeatedly met with excavations, which furnished during life only a more or less faint imitation of such resonance, he found himself constrained to admit "imperfect and doubtful" varieties of pectoriloquy—obviously nothing more than simple bronchophony. But he does not seem to have been aware that most perfect pectoriloquy, as defined by himself, may occur where a solid mass, of medium size, is interposed between a main bronchus and the surface, and hence, under conditions, the most strongly opposed to those of excavation. Facts of the first class show that cavities may exist without pectoriloquy; facts of the second, prove that pectoriloquy may exist without them: hence, in his notions of the diagnostic force of the sign Laennec was indubitably wrong. But neither one nor the other class of facts disproves the reality of the specific propagation of articulate sounds to the ear, as described by Laennec.

250. In conformity, then, with the principle of recognising the individuality of every sound having special acoustic properties, I retain pectoriloquy,—merely, however, as a variety of simple bronchophony, under the title of *pectoriloquous*. It closely resembles the resonance heard over the larynx, and may exceed this in intensity; like the laryngeal voice, it appears to pass directly through the stethoscope into the observer's ear, and may throw the choncha, and even the neighbouring part of the skull, into more or less strong vibration. Limited generally to a small and accurately defined space, it may have a hollow and ringing character or not. Though, generally speaking, loud, this is a wholly unessential property of pectoriloquous bronchophony, depending in great measure on the power of the laryngeal

voice: the hollow and ringing characters, the insulation of the phenomenon, and its transmission, in an articulated form, through the stethoscope, may be distinctly marked, even when the ordinary voice is almost destroyed. When the physical conditions of its production exist in a patient thus reduced almost to a state of aphonia, it becomes peculiarly characteristic,—low muffled whispers pass directly into the ear, articulated sometimes with as much, if not more, precision than the laryngeal voice (whispering pectoriloquy, *or whispering chest-speech*): here there is no tactile fremitus on the surface, nor is any thrill communicated to the choncha of the listener,—proof unmistakeable that such thrill is not an essential element of pectoriloquous bronchophony. The loud pectoriloquy of a cavity may in each syllable be followed by a sort of whispering echo.

251. Bronchophony becomes pectoriloquous in certain conditions of excavation in the lungs, and in cases where solid masses lie between the bronchi and the parietes. If the quality of the resonance be markedly hollow and ringing, and if it exist in the *whispering* form, I believe, notwithstanding the statements made to the contrary, that it strongly indicates a cavity: at least, I have met with no exceptional case. On the other hand, the most marked pectoriloquy of the *loud* form, without hollow and ringing character, I have almost ever heard, existed, near the right sterno-clavicular angle, over a fibrous nodule in the pleura,—the lung being healthy and simply slightly condensed at the spot by pressure.

Whatever be the nature of the excavation, gangrenous, purulent, apoplectic, cancerous, or tuberculous, globular dilatation of a bronchus included, pectoriloquy *may* be perceptible. But it may not occur at all; or, if occurring, may be transitory or intermittent. The conditions of an excavation most conducive to such resonance are,—moderate size; smoothness and density of its internal surface, hence absence of bands either traversing its area or coasting its walls; emptiness; superficial position, and especially adhesion of its periphery to the parietes of the chest; thinness and hardness of that portion of its walls next the surface; and free communication with the bronchi. Where, on the contrary, a cavity is possessed of flaccid irregular walls, is more or less nearly filled with fluid, and deeply seated, with healthy lung interposed between it and the surface, the resonance will be wholly deficient in pectoriloquous character, and may be strongly or faintly bronchophonic, or *absolutely null.* Moderate size is of importance; small cavities, unless under special circumstances of seat, are rarely pectoriloquous; and very large dimensions are equally opposed to such resonance.* Very small diameter of the communicating bronchi impairs the distinctness of the phenomenon; and want of communication with the bronchi, also, will prevent its development, persistently or temporarily, according as the obstruction is itself permanent or dependent upon passing circumstances, such as accumulation of sputa in their interior. On the other hand, as observed by Laennec, where the number of fistulous openings by which a large excavation communicates with the bronchi increases, pectoriloquy becomes indistinct or ceases altogether; and if a communication be set up between a cavity and the pleura, or if the contents of the former escape into the subcutaneous cellular membrane, the phenomenon of pectoriloquy disappears. It follows very clearly from these facts, that pectoriloquous bronchophony must be frequently wanting in cases of caverns in the lungs, and that the other signs of destruction of pulmonary substance are much more trustworthy.

252. When the vocal resonance has a metallic character, is not transmitted forcibly through the stethoscope, is not articulate, but conveys the impression of its being produced in a hollow space of large size, it is called *amphoric,* from the similarity of the phenomenon to that produced by speaking into an empty pitcher.

253. *Ægophony* (*αιγος*, gen. of *αιξ*, *a goat*, and *φωνή*, *voice*)

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* Thus (Green, U. C. H., Females, vol. v. p. 148), over an enormous tuberculous cavity furnishing perfect amphoric respiration with metallic echo, there was occasionally complete absence of vocal resonance of any kind.

the name given by Laennec to a special resonance, distinguished by its tremulous, nasal, and cracked character, suggestive of the bleating of a goat, is another variety of bronchophony. When most strongly marked it is distinctly ringing, jarring, and muffled; is synchronous with the articulation of each word, or follows it immediately, like a feeble, sometimes whispered, echo of higher pitch than itself; conveys the idea of somewhat distant origin; does not appear to traverse the stethoscope, but rather to flutter tremulously about the applied end; is commonly persistent, but of short duration; audible over a very limited surface, and occasionally capable of being altered in position by varying the posture of the patient. Certain modifications of pure ægophony have been happily compared by Laennec to the voice passing through a metallic tube or cleft reed,—that of a person speaking with a counter between his lips and teeth,—the nasal twang of the exhibitors of Punch.

254. Rarely lasting more than from two to five days, Laennec has known ægophony continue in cases of chronic pleuritic effusion for several months. It does not accompany all notes of the voice, nor all words, even though pronounced with the same pitch: this peculiarity does not depend on the loudness of the laryngeal voice.

255. Pure ægophony is observed in certain cases, where a stratum of fluid contained in the pleura compresses the lung. The precise thickness of the layer of fluid most favourable to its production cannot be either laid down as matter of observation, or satisfactorily calculated. Laennec states that he has discovered this sign, when there were not more than three or four ounces of fluid in the chest. The ascertainable facts are as follow:—At the earliest period of pleuritic effusion, when it seems deducible from physical principles that the fluid must be tolerably equally spread over the pulmonary surface, there is commonly rather a tendency to ægophony than actual ægophony. The lung is then —the inference arises—too slightly condensed, and the liquid accumulated too small in amount for the production of the

phenomenon. It appears in fullest force during the period of gravitation, before any detrusion of the parietes has occurred, and consequently while the fluid is still moderate in quantity, and at its upper part spread thinly over the pulmonary surface. With the increase of effusion it disappears altogether, to return again when absorption has reduced the liquid to a thin layer. So, too, in cases of paracentesis for empyema, ægophony sometimes appears after a certain portion of the fluid has escaped. On the other hand, exceptional instances occur, probably explicable by adhesions, in which ægophony remains in spite of very abundant accumulation. I have seen such cases, and such a one has been published by Andral (*Clin. Méd.*, t. ii., Obs. xxi.), where displacement of the diaphragm and heart gave evidence of the abundance of the fluid.

Supposing the patient to be examined in the sitting posture, the seat of the phenomenon will be found to be the neighbourhood of the inferior angle of either scapula, and a few inches on either side in a line with that angle; in very rare cases extending almost to the nipple in front. This limitation of seat is important as diagnostic of true ægophony. The shrill ægophonic quality of resonance, traceable to a naturally shrill and tremulous character of the voice would be thus at once distinguished from resonance actually caused by the presence of fluid in the pleura, inasmuch as it would exist in the highest degree wherever the dulness under percussion was most marked; that is, commonly at the base of the lung. Now ægophony does not exist where the dulness is greatest: far from this; such a quantity of fluid as is capable of causing very notable dulness will almost inevitably, as just seen, cause the disappearance of ægophony, if it have previously existed. Exceptional cases are met with, however, in which the seat of true ægophony is more extensive. Laennec sometimes observed it over the entire affected side at the commencement of the disease. In two such cases he "ascertained, by examination after death, that this peculiarity depended upon the retention of the lung in partial apposition with the chest by means of pretty numerous adhesions, so that the lung became invested by a thin layer of fluid over its whole surface. In cases of this kind, the sign in question is observable during the whole period of the disease." It has been matter of inquiry whether ægophony is produced precisely on the level of the upper border of the pleural fluid, or at a variable elevation of that fluid, where it is of a certain thickness. The point is a difficult one to decide; but the conditions of the respiratory murmurs and of the percussion sound appear, as a rule, to support the latter, the less commonly received, view.

256. Moveableness of ægophony is essentially a character of short duration: the displacement of the fluid either ceases to be possible from the interference of plastic exudation, or the quantity of fluid increases to such a degree as to exclude altogether the conditions of its development.

257. Inflammatory and dropsical accumulations of fluid in the pleura are the states to which true ægophony is almost peculiar, —its existence in cases of hydropericardium is altogether exceptional. In cases of pleuro-pneumonia, when fluid exists to the necessary amount in the pleura, the resonance of hepatisation becomes modified very usually by an ægophonic twang; but it is extremely rare to observe marked ægophony in these cases. It is true that, in the time of Laennec, the apparently important discovery was made that ægophony exists in some instances of simple hepatisation; but the observation appears to me hardly a correct one. I have never yet detected ægophony of even tolerably pure character, as an attendant on simple inflammatory induration of the lung, unless the ordinary voice of the patient was of shrill tremulous character. Hence this exceptional species of resonance is most frequently encountered in persons, more especially women, of advanced age. I have been gratified by finding that M. Grisolle's experience (*Pneumonie*, p. 242) has led him to a very similar conclusion. The bronchophony of hepatisation may, sometimes, be given an ægophonic character by directing the patient to speak with the nostrils closed.

258. The theory of vocal resonance, in itself of inferior clinical importance, has acquired great interest from the part it has been ingeniously made to play by Skoda in his attempt to remodel the doctrines of auscultation at large.

259. Laennec regarded bronchophony as an essential dependence on increased density of the pulmonary texture, and supposed that it was simply produced by the greater facility with which comparatively dense homogeneous tissue—homogeneous from the exclusion of air—conducted the laryngeal vibrations.

This explanation is inadequate to meet all the circumstances of the case. In the first place, bronchophony may exist, and this to an intense amount, over lung rarefied to such a degree as to give actually almost tympanitic resonance under percussion. Of this fact, not generally known, or, at least, taught, I have observed a certain number of examples, where *post-mortem* examination left no doubt of the absence of any textural change in the lung except emphysema. In the second place, as was, I believe, first mentioned in print by Skoda, the voice resounds sometimes with greater intensity over the chest than over the larynx itself. In the third place, the pitch of the bronchophonic, differs sometimes distinctly from that of the laryngeal, voice. These facts, without disproving the partial correctness of Laennec's theory, show at least that it requires an addition of some kind to make it include all the varieties of the phenomenon.

260. Skoda, holding the general doctrine that the "varying conducting power of the healthy and diseased lung-substance cannot be taken as a basis of explanation of the phenomena of auscultation,"* specially opposes Laennec's views of the mechanism of bronchophony on the following grounds:—

(*a*) Bronchophony may in the course of a few minutes appear and disappear over hepatised lung, the other physical signs, especially the percussion-sound, having undergone no change. (*b*) When vocal resonance thus suddenly disappears, it may as suddenly be restored by making the patient cough or breathe deeply, so as to free the bronchi from fluid in the part of the organ auscultcd. (*c*) In cases of pleuritic effusion, the resonance grows weaker and weaker in proportion as the fluid increases; now as the lung grows more and more solid, the greater the quantity of pleural fluid compressing it, the reverse ought to be observed, were Laennec's doctrine of solidification and improved conducting capability well founded. (*d*) If a healthy and a hepatised lung be removed from the body, and if, while one person speaks through a stethoscope placed in contact with the surface of each organ successively, a second listens through another stethoscope placed at an opposite point of that surface, the listener will find that more intense resonance reaches the ear through the healthy than the diseased lung.

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* Zweite Auflage, p. 36. In the fourth edition (p. 37) this negation is limited to the "strength and purity of the voice."

261. What is the force of these objections? Let us examine them *seriatim*. (*a* and *b*) Since I first became acquainted with Skoda's statement concerning the appearance and disappearance of bronchophony in hepatisation, I have repeatedly endeavoured to produce artificially the change he describes. Now, as a rule, the experiment has wholly failed.* In some rare instances a very slight passing change in the force of resonance may, it is true, be perceived,—the question is, what is its cause? Skoda, assuming that the alleged temporary disappearance of bronchophony over hepatised tissue depends on passing obstruction of the tubes with fluid secretion, conceives he has found an argument against Laennec's doctrine of increased conducting power in hepatisation; for "were such increase of power real, it would be a matter of indifference, whether air or fluid were contained in the bronchial tubes." But is this the fact? Is it true that, on the doctrine of increased conducting power, it makes no difference, whether one portion of the series of conducting media consist of fluid or air? Obviously the *onus* of proving the fact, if it be a fact, rests with Skoda: meanwhile, we are justified in believing, that such partial variation in the composition of the conducting materials does exercise a certain influence on the general result; an influence quite capable of explaining the very slight variations in the intensity of bronchophony, that have fallen under my notice. Again: Let us suppose a case of hepatisation of a tolerably thick stratum of the posterior part of a lung; bronchophony is heard, in such a case, at the posterior, not at the anterior, surface of the chest. Now, as, according to Skoda himself, reinforcement of voice would, under the circumstances, take place about the central tubes, how comes it, if his notion of the superiority of healthy over solidified tissue, as a conducting material, be constantly correct, that bronchophony is not heard in front over healthy texture, instead of being, as it is, audible in the back, over the hepatised parts. (*c*) The argument derived from the phenomena of pleuritic effusion seems feeble, and, indeed, unsound. The interposition of a mass of fluid between the condensed lung and the surface alters the terms of the problem completely. It has been shown by Colladon and Sturm, that sonorous rays which reach the surface of water at a very acute angle do not pass into the air, but undergo reflection in the interior of the liquid. Now the angle at which the sonorous vibrations reach the fluid from the bronchi, and ultimately reach the outer surface of the pleural fluid, may very possibly often prove of the degree of acuteness fitted to prevent their passage into the air. At all events Skoda, by ignoring the new influences likely to be exercised by the fluid, renders his argument valueless. (*d*) The results I have obtained from some experiments on the conducting powers of

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* E. g., Shay, U. C. H., Females, vol. ix. 144. (Pleuro-pneumonia) "Several coughs, which produce a good deal of loose rhonchus, do not in any way affect the characters of the bronchophony."

hepatised tissue, do not agree with those announced by Skoda. First: It is, I admit, quite true, that tissue, called hepatised, may conduct the voice no better, or even less forcibly, than a similar thickness of healthy parenchyma; but it is equally true, that this is not a constant result. I have occasionally found hepatised lungs, taken from the body, conduct the sound with extreme intensity. And these varying results may be obtained from different lungs, which the naked eye would judge to be in the *same* state physically, in regard of their shares of air, fluid, and semi-plastic substance: but it is evident, from the variations in result, that, acoustically, they are in *different* physical states; and that, therefore, such experiments as Skoda's are not to be trusted to. Specimens of parenchyma, apparently identical, are in reality widely different. There can, for example, be little doubt, that varying homogeneousness plays a more important part, than any observable so-called solidification, in regulating the conducting power of the lung. In the varying homogeneousness of different specimens may, in truth, lie the key to the difficulty; but even if so, it is a key which cannot practically be used. Secondly: If, while one person speaks into a stethoscope with its narrow end introduced into the trachea, a second listens over a part of the chest where hepatised lung lies beneath, and where intense sniffling bronchophony existed during life, the listener will often be surprised at the singular and total absence of sound. Skoda, admitting this fact, attempts to evade its force by supposing the vibrations to be interfered with by fluid in the bronchi. To this I would reply, that I have satisfied myself of the total absence of such *post-mortem* resonance over pneumonic solidification, in a case where the bronchi, to the third and fourth divisions, were peculiarly free from fluid, and scarcely any spumous liquid infiltrated the parenchyma—which very same parenchyma, removed from the body, conducted the voice from one stethoscope through another with striking intensity. If we consider the main difference in the physical conditions of the parts, when an individual himself speaks, or when another speaks into his trachea after his death, an obvious explanation of the experimental failures to imitate the bronchophony of life suggests itself. In the dead body, in truth, the laryngeal, tracheal, and bronchial walls take no share in the production or conduction of the sound, which is propagated by their contained air alone; whereas in life the walls of those tubes obviously conduct the sonorous vibrations. Besides, hepatised and healthy lungs are not strictly comparable in and out of the body in regard to this matter: *within the body* the contact of a hepatised lung with the chest-wall is more perfect than of a healthy one; and, admitting that the former is a worse conductor in regard of the condition of its substance, it may be a much better one through the closeness of its union, especially if adhesive, with the parietes. Here is a point which has been totally overlooked by the Viennese physician and his followers.

262. I conceive, then, that, whether Laennec's doctrine be true or false, the arguments just reviewed fail to prove it unsound. Skoda himself naturally

thinks otherwise ; and, excluding the walls of the trachea and bronchi from all share in the *conduction* of the sonorous vibrations of the chordæ vocales,* (an office assigned by him to the contained air of those tubes alone,) maintains that bronchophony is really produced by consonance of the air in the bronchial tubes with the laryngeal voice.

263. The hypothesis of consonance does not appear to me satisfactory ; and it unquestionably fails to meet all the circumstances of the case. The reasons on which I ground this opinion are as follow :—(*a*) Air in any enclosed space does not consonate with every sound produced at its orifice, but only with the fundamental note of that space, and with certain others having a fixed harmonic relationship to that fundamental note,—with certain of its *concords,* in short. This is easily ascertained, in a rough way, by running the gamut with the voice at the mouth of an empty water-bottle ; one note only of the octave is at all markedly reinforced by consonance within the cavity,—one or two others, (according to the distance from the orifice at which the vocal sound is emitted, and the depth of the mass of air within the bottle,) very slightly increased in loudness. Now, on the contrary, when bronchophony exists, it is audible with *successive notes* of the octave, standing in no harmonical relationship to each other,—absolute *discords,* in short. These successive notes are most loudly bronchophonic at the lowest part of each register, (whether bass, tenor, or soprano ;) but the force of bronchophony gradually decreases, not at harmonic intervals, but on *each successive note from below upwards,* until the resonance disappears altogether ; and obviously the greater force of bronchophony with grave tones, as contra-distinguished to acute ones, has nothing to do with the principle of consonance,—for consonance, where its conditions are fulfilled, will occur with notes of the latter, as well as of the former class. (*b*) Bodies consonate only in unison, or in certain fixed harmony, with the original sound which throws them into vibration.† Now the pitch of the bronchophonic voice varies irregularly from that of the laryngeal with which it co-exists. This difference of pitch is especially to be caught in cases of hepatisation, and is sometimes very striking in amount ; the corresponding notes heard in the larynx and on the surface of the chest are then, very perceptibly, discords. (*c*) Skoda's exclusion of the tracheal and bronchial walls from participation in the conduction of the laryngeal voice is at variance both with theory and experiment, and cannot for a moment be acceded to.

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* Auscultation ; Vierte Auflage, pp. 36, 40, and 68. He of course, admits, that the bronchial walls, in the spot where consonance takes place, intensify the consonating tones of the air within them by their own vibration ; but this is a very different thing from their taking part in the production of bronchophony by conducting the laryngeal voice.

(†) The unison-note alone is distinct to ordinary ears ; the consonating harmonics are so faint as to require the organ of a Costa for their detection.

(*d*) In cases where the bronchophonic voice is very positively and notably louder than the laryngeal, it is difficult to believe, from the mere fact of the intensity of the sound, that the phenomenon can be due to consonance. For a consonating sound, as a rule, is vastly more feeble than the primitive tone eliciting it: and the nicest adjustment of the quantity of air in the consonating body, presuming this to be hollow, is required, in order to produce any serious increase in the amount of loudness. Let it, however, be granted, *argumenti gratiâ*, that chance may sometimes cause the column of air between the larynx and the seat of bronchophony to be of the appropriate length to produce a marked increase of sound, —the doctrine of Skoda gains nothing by the concession. For, be it remembered, Skoda rejects conduction as an element of bronchophony,—bronchophonic voice is, in his apprehension, consonating voice alone; consonating voice is, then, under the above circumstances, by admission, louder than the original voice. Now, here is an idea irreconcileable with observation; for it does not appear, that (provided the original and consonating sounds be produced by bodies of the same class, as vibrating strings, hollow boxes, solid plates, &c.) the consonating sound is ever louder than the original tone.* (*e*) If the excess of loudness of the bronchophonic over the laryngeal voice were from consonance, *vocal fremitus* (inasmuch as the walls of the consonating tubes must vibrate in the direct ratio of the vibration of their contained air) ought to rise and fall exactly as *vocal resonance*. Now, as is notorious, these two phenomena do not invariably maintain any direct relationship to each other; one may gain, while the other loses, in intensity.

These objections appear to me conclusive against the pure doctrine of consonance, while they show that, if consonance plays any part in the production of bronchophony, it must be a subsidiary, rare, and accidental one. And even this concession is rather made on the ground, that the occurrence of consonance within the chest is, *à priori*, possible, than in deference to the arguments actually adduced in its favour.

264. From the discussion into which I have now entered, it would follow

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* The case may be very different where vibrations are communicated from a body of one physical constitution to a consonating body of another class. Thus, where a tube takes up the vibrations of a solid disc, the consonating note of the tube may (by managing carefully the length of the vibrating column of air in the interior) be made incomparably more powerful than the original tone: the quality of such notes is exquisitely pure and full; and long ago Savart suggested the construction of a musical instrument on this principle which, it seemed probable, would exceed in melody and power any of those in use. Civilised musicians have not profited by the philosopher's suggestion; yet the savages of some of the Pacific islands, curiously enough, have hit upon rude contrivances illustrating the principle.

that the mechanism of bronchophony is probably complex, and certainly not, as the attempt is commonly made to prove it, invariably one and the same.

265. There are four points which appear especially worthy of consideration: the conduction of laryngeal voice; its possible increase of intensity, within the chest; the distance at which that increase of intensity, if real, occurs from the part of the thoracic surface examined; and the relationship of pitch of the laryngeal and bronchophonic voices.

266. *First.* In regard of conduction of laryngeal sound, theory would support the inference, that as the human voice is best propagated in air, the more the lungs were rarefied, the greater would their conducting power become; and in accordance with this, it is certain that intense bronchophony is sometimes heard over highly emphysematous tissue. However, on the other hand, as the tracheal and bronchial walls themselves vibrate during speaking, any really solid material directly connecting a large bronchus with the surface of the chest, must conduct those vibrations forcibly; and, in accordance with this, we find that wherever solid fibrous structure is seated in the manner supposed, bronchophony of the most intense character is audible. But if the union of the solid material with the chest-wall be imperfect, if there be any interruption at the planes of union of the conducting materials, the acoustic conditions are completely changed, inasmuch as interruption at the union of media of different densities most deeply impairs the conducting faculty of the series. Here is one clue to the differences of vocal resonance, observed in cases where the physical conditions appear, on superficial view, identical; hence, too, we have no fair reason to expect that in all samples of the variable semi-solid states comprised under the title of "hepatisation," conducting power should be affected in an uniform manner,—as experiment shows that it actually is not. Experiment, in truth, alone can teach in each instance what the force of conduction really is in the various complicated conditions of physical change in the lungs.

267. *Secondly.* It is indubitable, that the bronchophonic voice is sometimes louder than that transmitted through the stethoscope directly from the larynx. The extreme rarity of this occurrence does not affect its reality; and hence, some explanation must be found for the increase of intensity within the thorax.

There seem to be three ways, as far as now known, in which a sound may be reinforced beyond the seat of its production;—by, what may be called, *unison-resonance,* by *consonance,* and by *echo.* In all three, reflection of sound is concerned; but the laws of that reflection are in each case different. This will be best understood from a tabular view of the differences of the phenomena. The reader will bear in mind that by unison-resonance is meant the reinforcement which occurs in the box of the guitar or violin when notes are produced from their strings, or when a musical-box, instead of being held in the air, is placed on a table: by consonance is understood the reproduction of certain notes of instruments or of the voice by other instruments, standing by: by echo is meant the well-known phenomenon of repetition of sounds.* All three agree in that the reinforced sound may exceed in intensity the original, and differ from this in quality.

Original and Secondary Sounds, how connected in regard of—

	Place of production.	Pitch.	Number of repetitions.	Number of Notes *coëtaneously* reinforced.	Time of production.
In unison-resonance.	Directly connected.	All notes of the octave intensified in unison.	No true repetition; only swelling of original sound.	Never but one.	Both simultaneous.
In consonance.	Separate but near.	A single note only of the octave (or its harmonics) intensified: that note is the fundamental note of the consonating body.	Only one.	May be one, and certain of its harmonics.	Both nearly simultaneous.
In echo.	Separate and more or less distant.	Same note only.	May be several.	None.	One distinctly sequential to the other.

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* Strangely enough, Skoda's printed statements on the signification of "consonance" are not altogether free from apparent contradiction. Thus, in one place, he includes as examples of "consonance," both the reproduction of a note of the human voice by a guitar-string, and the strengthening of tone which a vibrating tuning-fork undergoes when held against a table, instead of being held in the air; that is, he includes under the term "consonance," both consonance and unison-resonance, as I have defined them above. Yet, in a later page, he tells us that "consonating" bodies only reproduce their own fundamental tones, and certain others numerically related

268. Now the loudness of the bronchophonic voice is in all probability partly due, under different conditions, to each of these three agencies in turn.

There can be little doubt that, in the natural condition of the chest, the principle of *unison-resonance* comes into play. But it is totally unavailable in the explanation of the bronchophony of hepatisation, for the simple reason, that such *unison-resonance* is perfect, exactly in proportion to the *amount of air contained in the resounding space.* Imagine the box of the violin filled with any solid or fluid material, and what comes of its property of reinforcing sound?* Had this principle any influence in the generation of the bronchophony of pneumonia, its effect would obviously be to transfer the phenomenon to the healthy side of the chest, in cases where the solidification was limited to one lung.

Nor am I disposed to question that under peculiar circumstances there may be a repetition of sound by consonance, though this would be very difficult to prove: it does not appear to take place in hepatisation.

Echo, too, seems a very probable cause of reinforcement, and this in hepatised lung. The necessary conditions of reflection appear to be fulfilled: the tubes along which the voice is trasmitted from the larynx are surrounded by semi-solid material, proper, when compared with healthy tissue, to reflect and concentrate the sound; while the air-cells and minute bronchi are closed to a variable distance, and prevent its divergence. The tubes resemble so many speaking-trumpets, and, just as in these instruments, the augmentation of sound is produced by reflection from their quivering walls; as this reflection tends to propagate vibrations (otherwise divergent) in the same direction, increased intensity of sound must be the result. And, further, if the reflected vibrations chance to be brought to a focus within a large tube, then *echo* will occur; and, as under ordinary circumstances, the echo may be materially louder than the original sound. But it may be inquired, how, upon this theory, is the admitted temporary diminution of bronchophony explicable? Conceivably, by the deadening influence of accumulated fluid in the tubes.†

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thereto,—that is, he excludes unison-resonance, and hence, by implication, defines the phenomenon altogether differently (Vierte Auflage, pp. 37, 39).

* Every schoolboy knows the increase of tone given to the sounds of the Jew's-harp by placing it in the ordinary position for playing, within reach of a resounding cavity,—the mouth; and there are few who have not accidentally learned the hopelessness of attempting to play, if the said cavity happen to be partly filled with eatables.

† The effect of carpeting or woollen cloth of any kind, in deadening the sound of music in an apartment is well known. The intermixture of air and solid fibres in the carpets, through which the sound has to pass, deadens the echo between the ceiling and floor, by which the original sound is swelled.—Herschell, Art. "*Sound,*" *Encyc. Metrop.* Aerated mucus and sanguineous

Under these circumstances, bronchophony would, probably, (I have not verified this conjecture,) be heard at some point of the chest nearer the bifurcation of the trachea. Again, it is possible that certain changes of posture altering the relationship of the reflecting surfaces, might interfere with the production of echo, by preventing the reflected sounds from coming to their usual focus. Besides, the position of the auscultator in respect of the focal point might prevent him from hearing an echo really existing.* The force of the echo will also rise, the smoother the bronchial walls, and the larger the tubes in which it occurs. And numerous other circumstances may be conceived, but scarcely proved, to affect the phenomenon. Among these, the composition of the gases within the bronchi may, for aught that is known, hold an important place: hydrogen has been proved to deaden sound greatly; the effect of carbonic acid, mixed with other gases and aqueous vapour, can only be ascertained from experiment.

269. *Thirdly.* As concerns the distance from the point of auscultation at which the reinforcement of sound within the thorax occurs; the further away, the less of the resonance will reach the surface: the amount, however, will be modified by the conducting property of the interposed media.

A little consideration will show that these three conditions of bronchophony,—conduction of laryngeal sound, increased intensity of this within the thorax, and proximity of site of the increase,—may or may not be directly as each other; one may be in a state favourable to, the rest unfavourable to, the formation of bronchophony. Hence the variable state of the sign in different cases of the same disease.

270. *Fourthly.* The relationship of pitch of the bronchophonic and laryngeal voices seems the most difficult part of the subject,—difficult, at least, in those cases where a distinct difference can be detected in the pitch of the two.

That the voice should, in travelling from one spot to another, undergo alteration in pitch, seems, on first thought, opposed to the commonest experience and to the recognised laws of acoustics. And in experimentally investigating the point, there are some easy sources of fallacy. Bronchophonic voice may be muffled and husky, while the laryngeal tone is pure; and the

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serum in the bronchi would have the same effect on vocal echo in those tubes, as the carpeting under the circumstances referred to above.

* The existing theory of echoes generally is inadequate to explain many of their phenomena. There is, or was, a ruined fortress near Louvain well illustrating this. Here, if a person sings, he only hears his own voice, without any repetition; those who stand at some distance hear the echo, but not the voice,—and they hear the echo with surprising variations,—sometimes louder, sometimes softer,—now near, now distant.—*Burrowes's Cyclop.*, Art. "*Acoustics.*"

quality of the two may be essentially unlike. Now these differences may readily, unless great care be taken, be confounded with differences in pitch. Fully alive, however, to the possibility of such deception, I have endeavoured to guard against it; and am persuaded, that the pitch of the bronchophonic voice does sometimes irregularly differ from that of the laryngeal. How, then, is the difference explicable? Conceivably (1) by the production of a new note within the chest chiming in with the laryngeal; or (2) by change of laryngeal note during conduction through *varying* media in the thorax.

(1) The production of new note within the chest is, we have seen, possible by unison-resonance, by consonance, and by echo. But the very name, *unison*-resonance, shows that this principle cannot be employed in explanation, when difference of pitch is concerned. Nor will the principle of consonance serve us either. For, though it is true that under favouring circumstances a note differing in pitch from the original one may be generated by consonance, that different note is always an harmonic of the original tone. Now, the difference of pitch we have under consideration is irregular and non-harmonic. Nor will echo help us through the difficulty; for, though an echoed sound may differ from its original in intensity and duration, and even in quality, it always agrees with it in pitch.

(2) We are driven, then, to the phenomena of conduction through varying media for release from our difficulty. And though it would probably be impossible to *prove* that the change in pitch is thus actually effected in the chest, there is strong argument in favour of this mode of agency.

Thus (it appears from an experiment of Savart) "let a long flat glass ruler or rod, connected with mastic to the edge of a large bell-glass, perpendicular to its circumference, be very lightly supported in a horizontal position on a bit of cork, and then let the bell-glass be set in vibration by a bow, at a point opposite the place where the rod meets it . . . In this combination *the original tone of the bell-glass is altered, and the note produced differs both from that yielded by it, or by the glass-rod vibrating alone.*"* Again, Odier long since ascertained, that if hydrogen be breathed, the voice is *raised in pitch.*†

Now, here are facts showing that pitch may be modified by conduction from one kind of vibrating solid to another, and that the pitch of sounds is controlled by the nature of the gases in which they are produced. The application of these facts to our subject is sufficiently obvious; and, curiously enough, the revelations they afford on the question of altered pitch, lend unexpected support to Laennec in assigning the importance he does to conduction [259].

271. Finally, bronchophony seems to be a resultant, in lung-consolidations, of conduction and echo: in emphysema, of conduction and unison-resonance:

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* Quoted by Herschell, Art. "*Sound*," p. 807, *Encyc. Metrop.*

† Eod. Loc., p. 766.

in lung-excavations, of conduction, unison-resonance and echo: in cases of tumor uniting a bronchus, or compressed pulmonary substance, to the surface, of conduction in the main, of unison-resonance in a secondary degree. Besides, when the necessary acoustic conditions exist,—that is, when the tones of the laryngeal voice chance to bear a certain mathematical relationship to the fundamental note of a resounding space in the chest,—true consonance may take a part in the production of bronchophony.

272. There are besides, some subsidiary conditions, the influence of which cannot be doubted; viz., the density of the gases in the thorax, their composition, their temperature, and the quantity of fluid in the tubes.

But, even with the aid of all these conditions, there are some peculiarities of voice-resonance inexplicable. How happens it, for instance, that, in the great majority of persons, the voice naturally resounds much more forcibly under the right than the left clavicle?

273. Ægophony, according to Laennec, is the natural resonance of the voice in the bronchial tubes, rendered distinct by the compression of the pulmonary texture, and tremulous by its transmission through a thin layer of fluid in a state of vibration. He thought it probable, also, that the flattening, which the bronchi undergo from pressure of the pleural fluid, had a good deal of influence in its production; the quality of resonance being such as might be anticipated from the shape of the vibrating tubes, resembling the mouth-piece of the bassoon and hautbois: it is not sufficient in itself for the production of the phenomenon; otherwise, ægophony would exist in cases of absorption with *contraction* of the chest, which is not the case.* Laennec adduces various arguments in favour of these views, and affirms that by applying a bladder half filled with water over the larynx, the natural resonance is transmitted through the liquid with heightened pitch and slightly tremulous character. Skoda, holding that pure ægophony is audible in cases of pneumonia, and tuberculous infiltration, with or without cavities, as perfectly as where fluid exists in the pleura, and maintaining that a piece of liver interposed between the larynx and stethoscope will produce the same effect on the transmitted voice as a stratum of water, rejects Laennec's doctrine *in toto*. He believes that the tremulous character arises only from impulses of a solid body against some other body, solid, fluid, or aeriform,—impulses which cannot occur within the chest, unless the voice consonates therein in a space filled with air; and that, "probably" in most cases, the wall of a bronchus, in which the air consonates, re-acts through impulses on that contained air, and so causes ægophony; while it is also possible that the peculiar character may be occasionally given by "mucus, &c." imperfectly closing the orifice of a bronchus, and imitating the reed in the mouthpiece of reed instruments. Dr. Sibson believes ægophony

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* At least during the early period of such absorption; uniform or globular dilatation of the bronchi is well known to ensue at the advanced periods.

to be "pectoral resonance accompanied by whispering friction-sound; the two sounds are heard together, just as the drone and the notes of the bagpipe."

274. Dr. Sibson's theory seems inadmissible; because pure ægophony may be heard without the least shadow of friction-sound accompanying respiration, and because in cases of hepatisation with slight plastic exudation on the pleural surface, there may be abundant friction-sound, while the vocal resonance is totally deficient in ægophonic quality. Besides, the chest-motion during speech is scarcely sufficient to produce friction-sound,—which, at all events, could only be expiratory in rhythm. If Skoda's main theory were well-founded—that of the quivering reaction of bronchial tubes on their contained air—it seems difficult to understand, why the bronchophony of hepatisation should not always be ægophonic; seeing that, as he himself teaches (what is of course true), vibration of the tubes always occurs, when their contained air consonates. The subsidiary suggestion concerning the imitation of reed-instruments by bronchial mucus is ingenious and plausible. Yet, whereas marked bronchophony with more or less liquid rhonchus sometimes exists over lung, at once emphysematous and bronchitic, I am not aware that an ægophonic quality has under the circumstances ever been detected: it may be rejoined, that the conditions of consonance do not exist here; but this would scarcely be a fair argument, as bronchophony (an alleged effort of consonance) does exist. For my own part, believing, as I do, that while other conditions may lead to a close simulation of ægophony, the pure quality described by Laennec depends upon the interposition of fluid, I look for the explanation of the phenomenon in some degree at least to that fluid. Now Laennec's experiment is too rude an imitation of the state of things in the pleura to be trusted to, though a slightly quivering character does very positively attend the resonance in the manner he affirms. The abruptness and the peculiar quality of ægophony are easily explicable by the intervention of liquid; the experiments of Colladon and Sturm have shown that the duration of sounds similarly produced differs notably in water and in air, and that their quality is completely different. Thus a bell struck under water gives no tone as in air, but a quick sharp sound, as of two knife-blades clashed against each other [261].*

275. Before concluding the subject of vocal resonance, it may be observed that efforts have been made to connect peculiarities in the resonance of the observer's own voice (as he speaks with the ear applied to the chest directly, or with the intervention of the stethoscope,) with the amount of density of the parts beneath. Few auscultators can have failed to notice that while their voices sound with strong vibration from some

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* Annales de Chimie et de Physique, t. xxxvi. pp. 243 and 254.

chests, or from certain parts of these, no such resonance occurs from others: it is greatly more marked when the solid, than the hollow, stethoscope is used. I have, however, not found any condition of lung uniformly attended by, or uniformly free from, this sort of resonance, called autophonia by Hourmann; nay more, it may be so strong over an *abdominal* tumor, as to cause pruritus of the ear applied to the stethoscope.* I consequently, in the present state of knowledge, attach to it no clinical value. I have known this resonance have an ægophonic quality in pleuritic effusion.

§ III.—Resonance of the Cough.

I.—IN HEALTH.

276. If the stethoscope be applied over the larynx or trachea of a healthy person while coughing, the act of expiration is found to be accompanied by a sound of hollow character, varying in respect of graveness and intensity with the voice of the individual; the observer is not conscious of any sensation of succussion in the site of its production. Auscultated on the surface of the chest, the cough in health furnishes a quick, short, commonly dull and indistinct, somewhat diffused sound, produced at a distance, without hollow or tubular character, not attended with any distinct sensation of succussion in the interior of the thorax.

II.—IN DISEASE.

277. *The modified states* of the pulmonary cough, which occur in disease, are the *bronchial, cavernous, amphoric.*

278. *Bronchial cough*, when well marked, is a sound of harsh character; is attended with a sensation of very marked succussion in the chest, and a slight degree of impulsion towards the ear of the observer; is very rapidly evolved, and more concentrated under the instrument than the natural sound.

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* Dujardin, Enlarged Spleen, U. C. H., Males, vol. v. p. 198.

279. *Cavernous cough* is characterised by its perfect hollowness and metallic character. The sensation of production in an excavated space of limited size, the strong impulsion and transmission of the sound through the stethoscope with a force sometimes painful to the ear, are quite distinctive of this species of resonance. Cavernous cough may be pure, or associated with cavernous rhonchus; if fluid be present in the cavity to a moderate amount, it will not interfere with the production of the characteristic cough, and the forcible agitation the liquid matter undergoes during cough will of course be attended with rhonchus.

280. *Amphoric cough* is a loud resounding sound of metallic character, conveying the notion of production in a large space more or less empty; it is not forcibly transmitted through the stethoscope.

281. The varieties of thoracic cough are heard in the same cases as the corresponding varieties of respiration; they are of little utility in diagnosis. In pleuritic effusion, the quality of the cough is sometimes quasi-ægophonic.

§ IV.—PHENOMENA COMMON TO THE RESPIRATORY SOUNDS, TO RHONCHI, AND TO THE RESONANCE OF THE VOICE AND COUGH.

282. Differing from all the morbid conditions hitherto considered, the phenomena termed amphoric echo and metallic tinkling, attend the acts of breathing, of coughing, and of speaking. These phenomena are fundamentally one and the same,—the echo of various sounds, reflected by the walls of a capacious hollow space within the chest, under circumstances modifying the force, concentration, quality and pitch of that echo. Metallic tinkling is the term originally applied by Laennec to a clear, ringing, highly metallic, single sound, of very high pitch, not dissimilar to that produced by gently striking a hollow glass vessel of globular form with a pin. Its quality may, however, be more or less purely metallic, and its pitch fall, and its

clearness diminish, the tinkling character gradually disappearing *pari passu*, till it gradually merges in the low-pitched sound of buzzing amphoric echo. In different cases, or at different times in the same case, this transition may be detected: and there is a certain stage of the transition in which it is difficult to determine whether the term metallic tinkling or amphoric echo be the most applicable. The metallic quality, though less clearly and sharply defined, is yet very obvious in amphoric echo,—which may be imitated with some success by speaking, breathing, or singing into an empty water-bottle.

283. Metallic tinkling, occurring in connection with respiration, co-exists commonly with (or rather, echo-like, follows), inspiration, being prolonged somewhat into the succeeding expiration; it is very rarely limited to the latter. Generally speaking, it alternates irregularly with an amphoric state of the respiratory sounds, the one unnatural state giving place to the other, after a variable and for a variable number of respirations. It appears to be produced either deep within the chest, or near the surface: and is rarely persistent for any considerable number of respirations. Amphoric echo may attend both sounds of respiration, or be limited to either.

284. Generally audible at the central height of the chest, laterally or posteriorly, (whence they may be propagated with gradually diminishing intensity to the surrounding parts,) metallic tinkling and amphoric echo may be heard in every part of the thorax. The most clearly marked and intensely developed metallic tinkling I ever heard, was chiefly audible under, and a little outside, the nipple: the case was one of tuberculous perforation of the pleura.

285. The mechanism of metallic tinkling and amphoric echo has been long sought after with all the eagerness of curiosity; but observers are far from having come to a uniform conclusion on the subject. I believe, as just mentioned, that the two phenomena are one and the same, fundamentally,—echoes of different properties from the walls of a large space, more or

ess favourably disposed for reflection and concentration of ounds, produced either within the area, at the outlet, or in he close vicinity, of that space. It appears, too, that the low-itched buzzing echo only requires the presence of air in the ollow space, though water, in moderate proportional quantity, may be present therein; while the high-pitched tinkle requires fluid for its production,—not that such tinkle is physically impossible unless fluid be present, but that in the chest, the conditions, independent of fluid, which are capable of generating it, do not coexist. In experimental support of this statement, it may be observed that if we blow, cough, speak, or sing, into an empty glass decanter, a low-pitched, buzzing, amphoric echo only will be produced; metallic and ringing in quality, it is true, but never of the tinkling pitch.* Let a little water now be placed in the decanter, and the result will be exactly the same, so long as the fluid is not agitated. But agitation of the fluid changes the character of the echo. Thus, let drops of water, slowly, and at distinct intervals, fall on the surface of the fluid in the decanter, and the ear, applied to the surface of the vessel, recognises the most perfect imitation of metallic tinkling; just as in certain instances the phenomenon occurs within the chest, independently of respiration, rhonchus, voice, or cough, when a patient, with a very large cavity, or with hydro-pneumothorax, suddenly changes from the recumbent to the sitting or erect posture, and when, in all probability, a drop of fluid is precipitated from the roof of the cavity to the fluid on its floor. Or, again, breathe into the water by an elastic tube, and the bubbling will be found to produce a perfect tinkle.† It is probable, too, that sounds, generated in fluid, on the close confines of a cavity, itself free from fluid, may be echoed with metallic tinkle by that cavity. Metallic tinkle seems to be the echo of a bubble, or at least of a sound generated within liquid.

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* The fundamental and consonating note of such vessels is always of low pitch.

† The experiments of Dr. Bigelow (Brit. and For. Med. Rev., vol. vii. p. 569), made post-mortem on a patient with hydro-pneumothorax, gave similar results.

286. The morbid states in which these phenomena have been observed, are hydro-pneumothorax with and without bronchial communication, simple pneumothorax, and large tuberculous excavations in the lung-substance. In the first case, where the pleural cavity contains air and liquid, and opens into the lung, both kinds of echo may occur with respiration, rhonchi, speech, and cough. If respiration produce in any way single, isolated bubble-sounds, either by the bronchial fistula opening below the level of the liquid or otherwise, metallic tinkling will occur; if there be no bubble-sound, amphoric echo only will be heard: hence, if the fistula open above the level of the liquid, there will be amphoric echo, unless the fluid be by some means or other simultaneously agitated. Rhonchi produced in the communicating bronchial tube or tubes, will be echoed with tinkle, if their component bubbles be separately, and, as it were, intermittently evolved; if otherwise, amphoric echo will be the result. The influence of speech or cough will similarly vary. Fournet has endeavoured to show that the occurrence of one or the other variety of metallic sound, tinkling or amphoric, will also be found to depend upon the freedom and rapidity with which the escape of air through the fistula occurs. If it make its way from the fistula by rare, slow, and successive bubbles, tinkling will be evolved; if the bubbles be numerous and closely following each other, amphoric echo will be the result. This idea seems to me well founded; if the drops of water be allowed to fall rapidly into the decanter, in the experiment I have already referred to, the sharp tinkle passes into the low-pitched and confused amphoric echo. Fournet further holds that (as the level of the fluid is in some cases capable of being changed with the position of the chest, and hence the relation of the fistulous opening to that level altered), the same opening may at one time be the possible source of metallic tinkling, at

another, of amphoric echo. Again, if the size of the opening increase much, amphoric echo will take the place of tinkling; and *vice versâ*, if its caliber be diminished by obstruction with pseudo-membrane or otherwise. Both phenomena will cease, he holds, if complete closure of the opening be effected. It is, however, matter of certainty that either kind of echo may occur independently of communication between the pleura and bronchi; this last statement is consequently incorrect. In the second case of non-fistulous hydro-pneumothorax, the agitation of the fluid by coughing or by movement, or the fall of drops of fluid from the upper to the lower parts of the pleural cavity, and perhaps even the echo of rhonchi in the adjacent bronchial tubes, will cause tinkling. Thirdly, metallic echo, and even tinkling, both vocal and tussive, we are assured by some observers, have been heard in cases of simple pneumothorax, in which there was neither liquid effusion, nor perforation of the pleura. Low-pitched echo may intelligibly be produced under the circumstances; but the absence of fluid, especially where vocal sound is the alleged cause of the phenomena, makes it desirable that, in respect of tinkling, the observation should be repeated. On the rarity of simple pneumothorax it is needless to insist. Fourthly, large dimensions are an essential character of pulmonary cavities, that give either variety of metallic echo: all the four modes of production of both varieties may come into play within them.

287. That the metallic phenomena should be, as is the fact, best heard in connection with coughing and speaking, is just what might have been anticipated: these acts require greater force of respiration than ordinary breathing; they are, therefore, on the one hand, capable of propelling air through a passage which would have resisted its progress under a less impulsion; and, on the other, they themselves being more sonorous, more readily lead to audible echo. Forcible and deep respiration will produce somewhat similar effects.

§ V.—SOUNDS AND MURMURS OF THE HEART, AS TRANSMITTED THROUGH THE SUBSTANCE OF THE LUNGS.

288. In order to avoid repetition, I must refer the reader to the Second Chapter of this Part for an account of the mode and extent of propagation of the heart's sounds in the normal state of the thoracic organs: there, too, are described the changes in transmission of its sounds produced by disease of the heart itself and of the great vessels. We have here only to do with cases where, the heart and great vessels being healthy, morbid states of the lung and its appendages, by changing the conducting power of the media intervening between that organ and the surface where auscultation is performed, pervert the natural mode of propagation. Now, inasmuch as the physical sources of sound in the heart are fluid and solid only, and aeriform matter has no direct connection with them, the displacement of air in the lung, either by fluid or by solid matter, would be favourable in theory to the conduction of the cardiac sounds through the pulmonary substance: while increase of air within the thorax would have the contrary effect. And so the positive intensity of sound produced in the heart remaining unaltered, its relative intensity, as discovered at different parts of the thoracic surface, might be changed.

289. Experience supporting, in the main, this theoretical consideration, teaches us that whenever the cardiac sounds (the heart itself, the great vessels, and chest-walls being healthy), are found to be of greater intensity at any given point of the thorax, than at some other point nearer that organ, the lung, pleura, or mediastina have either in the former situation undergone some change, rendering them unusually good conductors of sound, or, in the latter situation, undergone some alteration diminishing their conducting power. The anatomical state in the first class of cases will be one of condensation or induration; in the second, of rarefaction.

290. Increased intensity of transmission of the heart's sounds is thus observable in pneumonia, chronic pulmonary consolidation, tuberculous disease, extensive pulmonary apoplexy, and œdema, dilatation of the bronchi, cancer of the lung, and solid accumulation in the pleura or mediastinum. The influence of pleuritic effusion will vary with its amount; the sounds will be better heard through a small extent of badly-conducting lung, than through a large mass of better-conducting fluid. Hitherto phthisis has been almost the only affection in which this means of diagnosis has been commonly applied. If the heart's sounds be more distinctly audible under the right than the left clavicle, and if the excess be sufficiently marked to leave no doubt as to its reality in the mind of the observer, the circumstance, in conjunction with the locality of its existence, affords presumptive evidence of tuberculisation. Generally speaking, other signs of a more direct character are observed at the same time; but in certain cases of incipient and rather deep-seated tuberculous deposition, it is often a source of satisfaction to have this additional sign to apply to. Its absence would not, however, by any means impugn positive evidence of consolidation derived from other sources. Whatever be the cause of the sign, it is for obvious reasons more readily substantiated at the right than the left side.

291. Diminished intensity of transmission of the heart's sounds, on the other hand, accompanies highly marked emphysema. The importance of this fact is habitually acknowledged in respect of the præcordial region, where the sounds may be almost completely muffled by an intervening thick mass of rarefied lung; but the sign may be established elsewhere. Thus, in a case of intense emphysema of the left lung, to which the disease was limited almost completely, and especially marked at the posterior aspect of the chest, I some years ago detected that the heart's sounds were considerably more distinct posteriorly on the right than the left side. As there was no evidence of induration of the right lung, and as the sounds there

were not louder than is sometimes observed in healthy individuals, the difference on the two sides could only be ascribed to diminished conducting power on the left. This sign, in the rare cases where it could be established, would appear to warrant the diagnosis of general emphysema of the substance of the lung in its deeper parts, as well as on its surface,—a point of some importance; for diagnosis, as well as anatomical investigation after death, generally aims too exclusively at the detection of superficial emphysema. The influence of pneumothorax varies; as a rule, it impairs the force of the sounds, but sometimes they seem to echo in the pleural cavity, as they do in a flatulent stomach, and so become intensified: I have observed this variation within twenty-four hours.

292. When the conducting influence is a weakening one, the observer should specially notice the second sound of the heart; when an intensifying one, the first.

293. Whether the heart's sounds may be modified in the course of transmission in virtue of any other principle than conduction, will be elsewhere considered [410].

294. The conduction of cardiac *murmurs* is modified on the same principle. But does any condition of lung generate either cardiac or vascular murmur, independently of disease of the heart, or of those conditions of the blood, spanæmic and other, which render its movement sonorous? It is by no means very uncommon, as Dr. Stokes was the first to state, to observe a sharp blowing murmur in the subclavian artery where the apex of the lung is consolidated by tubercle,—a murmur completely wanting in the heart, aorta, carotid, or opposite subclavian. Dr. Stokes ascribes it to falling in of the infra-clavicular region, to consolidation of the lung, and to sympathetic irritation. He has found it remittent occasionally, and removed by leeching or by an attack of hæmoptysis. My observations agree almost completely with those of Dr. Stokes. But the *nature* of the consolidation is a matter of indifference; and I think this murmur is of rare occurrence, unless there be systolic murmur

at the base of the heart. It is sometimes connected with a murmur at the second left, or pulmonary, cartilage, evidently seated in the artery of that name; is more common on the left than the right side; is greatly increased in force by suspension of the breath; sometimes disappears and reappears in the course of a few minutes; is sometimes removed by change from the sitting to the lying posture, and *vice versâ*, or even by brisk rotation of the arm. In quality the murmur varies from soft blowing to sharp whistling. It may continue for years, and seems more frequent in men than women. The pressure to which the vessel is submitted from the indurated and commonly contracted lung, especially when coupled with the least spanæmic tendency of the blood, seems its essential cause,—though the condition of the circulation in the part may impede or promote its formation.

295. Dr. Latham points out soft, blowing, systolic murmur, limited to the pulmonary artery, as a frequent concomitant of tubercles in the lungs. I have met with it occasionally unassociated with subclavian murmur; but of this more hereafter.

SECTION VI.—SUCCUSSION.

296. It has been seen that the succussion of certain contents of the chest, produced by the heart's impulse, and by the act of coughing, may give rise to physical phenomena of diagnostic import. And it was known to Hippocrates that if the chests of certain patients, labouring under thoracic diseases, be shaken, a "sound may be heard on the affected side." Hippocrates, however, erroneously supposed empyema to be the disease giving rise to this sound: his pathology was defective, but his observation correct: and the phenomenon retains to the present day the name of Hippocratic, or thoracic, succussion-sound.

297. The *succussion* necessary for the production and detection

of this phenomenon may be performed by pushing the patient's trunk abruptly, but with gentleness, forwards and backwards, while the observer's ear is applied to the chest; or the patient may himself move his chest once or twice in the manner indicated. The sound resembles closely that perceived on shaking a decanter, partly filled with water, close to the ear. Like that, it is a guggling splashing noise, the precise tone of which varies with the density of the fluid, and the proportional quantities of fluid and of air present. It differs in point of intensity according to the suddenness and force of succussion; but may be so easily produced as to be detected on the least movement of the patient, or during coughing. It may be audible at a distance from the chest, and be heard, while the splashing movement is felt, by the patient himself; and is, or is not, accompanied with metallic tinkling. Its duration varies greatly,—it may last for years, though this is very rare: in such chronic cases it is perceived by the patient, as he walks down stairs, rides on horseback, &c. It is not invariably a persistent condition when once developed; within twenty-four hours it may be present and cease to be producible, to recur again within a short period.

298. Produced by abrupt collision of air and liquid in an echoing place of large dimensions, the sound under consideration may be detected in hydro-pneumothorax, with or without bronchial fistula, and is occasionally to be heard in tuberculous excavations of unusually great size. Commonly audible over the general surface of the affected side, it may be limited to the anterior regions.* The sign is, however, by no means always to be discovered in hydro-pneumothorax: and one reason of its absence, thickness, and proportional excess of purulent fluid, was mentioned by Hippocrates:† it is certainly true that the thinner the liquid, the more readily is the sound produced.

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* Louis, Phthisie, éd. 2, p. 412. Paris, 1843.

† Laennec, by Forbes, Amer. edit. p. 541.

SECTION VII.—DETERMINATION OF THE SITUATION OF CONTIGUOUS PARTS AND ORGANS.

299. The object of attempting to determine the situation of other parts than the lungs themselves, when the diseases of these organs are the subject of investigation, is, as might be anticipated, to infer from any change in that situation the existence of some pulmonary affection capable of producing it. Experience has shown that such displacements do occur: and further, that when present they are among the most conclusive, as they often are the most readily ascertained, signs of the associated pulmonary affection.

300. The organs and parts liable to undergo displacement in consequence of pulmonary disease are—The Heart, the Mediastinum, the Diaphragm, the Liver, the Spleen, and the Stomach. The existence of displacement of these parts and organs is determined by means of various other methods of physical diagnosis,—by inspection, by application of the hand, by percussion, and by auscultation; very rarely by mensuration.

301. The Heart may be removed from its normal position by Detrusion, Elevation, and Procidentia.

302. *Lateral detrusion*, for obvious reasons more readily detected when occurring towards the right side, is there commonly associated with procidentia,—on the left, with some degree of detrusion, backwards and upwards. The progress of the displacement to the right side is usually gradual from its commencement till it has attained its greatest amount, when the organ pulsates between the fifth and seventh ribs to the right of the sternum. On the left it may be pushed almost under the axilla, its point being at the same time raised the width of an intercostal space, or thereabouts, and carried backwards towards the scapula.

303. Pleuritic effusion and hydro-pneumothorax are the

affections which drive the heart sidewards to the maximum amount; simple pneumothorax is a rare cause; hæmothorax is rarely copious enough; and hydrothorax, being generally double, does not displace the heart in this precise manner. Intra-thoracic tumors and aneurisms, variously placed, sometimes produce this effect; hypertrophy, as well as emphysema, of either lung, are among its occasional causes. Besides, the heart may be *drawn* as well as *pushed* sidewards—a mode of displacement that occurs in some cases of rapid absorption of pleuritic effusion, of consolidation with marked contraction of the substance of either, but especially the right, lung, of pure atrophy, and also of great diminution of bulk from tuberculous disease of the same organ.* The practical interest of this matter is connected almost solely with pleurisy.

304. The heart can scarcely be pushed forwards by any lung-affection, except emphysema; and various more prominent conditions, such as the distension of a thick stratum of lung in front of the organ, tend to mask this displacement. Intra-thoracic tumors and aortic aneurisms lying behind the heart, push it forwards, and, especially in the latter disease, give rise to very peculiar signs.

305. *Elevation* of the heart above its natural level, a displacement of very rare occurrence as a consequence of pulmonary disease, though sufficiently common in cases of abdominal tumor † and ascites, is sometimes seen as an effect of diminished bulk of the apex of the lung. Such diminution only occurs in

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* I have twice, at the Consumption and University College Hospitals, seen the heart permanently beating in the right thorax, where no pleuritic effusion on either side had ever existed, as far as could be made out by present signs or past history, where the liver and spleen lay in their natural positions, and where great tuberculous excavation and destruction on the right side seemed to have *drawn* the heart in that direction, aided by perhaps the *detruding* influence of hypertrophy of the left lung.

† I have known the heart permanently raised an intercostal space by the splenic enlargement of leucocythæmia. Case of Dujardin, U. C. H. Males, vol. v. p. 192. June 1850.

tuberculous disease, and is produced by atrophy of the lung-substance, closure of air-cells, and contraction of exudation-matter, both interstitial and pleural. I have never observed the apex raised higher by this cause than the fourth rib and third interspace; but in ascites, both in the male and female, and in ovarian dropsy, I have known it impossible to feel any impulse lower than the second interspace.

306. In *procidentia* of the heart the organ is below its natural level, and carried somewhat towards the median line; the impulse is then much more decided at the epigastrium, especially between the ensiform cartilage and left false ribs, than in the cardiac region. The common pulmonary cause of this displacement is double emphysema, of which it furnishes one of the most characteristic signs. It rarely exists to an appreciable extent in double bronchitis, if there be no emphysema; neither does double hydrothorax commonly induce it. The advance of tumors in certain situations may of course conceivably carry the heart downwards, but clinically this influence is rare.

307. The *Mediastinum*, at its lower part, is of course carried to the right or left by such morbid states of the lungs or pleura as produce lateral displacement of the heart. Superiorly, above the third rib, the mediastinum may be encroached upon by the lung, without any displacement of the heart, and be sometimes pushed more or less to the opposite side. Emphysema of either upper lobe will produce this effect, and if both organs are implicated, the mediastinum may be, as it were, obliterated by its pleural borders being brought into close juxta-position; the approximation of the edges of the lungs may take place so high as the sternal notch: the percussion-sound will of course be unnaturally clear. Tumor connected with the upper part of the lung, circumscribed empyema, tuberculous accumulation in the pleura, and acute hepatisation, may cause encroachment on the mediastinum, with dull percussion-sound: tuberculisation of the lung itself never, except perhaps quite at the outset, produces this effect, the disease tending to diminish the bulk of the

organ—hence occasionally a valuable aid in diagnosis. It is unnecessary almost to add that mediastinal tumors alter the relationships of the mediastinum; and that diseases of the great vessels, and of the heart likewise, deeply affect them.

308. *The Diaphragm.*—In the *normal* state, the upper edge of the arch of the diaphragm, reaches, in the adult, the level of the fourth interspace on the right side, that of the fifth rib on the left, while the central tendon lies a little lower than this. The right side of the chest is consequently somewhat shallower than the left. In children the entire diaphragm rises somewhat less within the thorax. Full eating, and flatulent distension of the abdomen, temporarily raise it somewhat. The influence of tight-lacing will vary with the fashion of the day: if the waist be "worn high," the constriction will depress, if "low," will raise, the diaphragm.

309. The position of the right wing is ascertainable by percussion of the liver anteriorly; where the sound becomes clear on forcible percussion, carried from below upwards, lies the upper border of the liver, and by inference, the convexity of the arch of the diaphragm. The cessation of vocal fremitus, where the liver is uncovered by lung, will corroborate the results of percussion, and supply a measure of the depth of liver overlapped by lung. The main guide to the position of the left wing will then be the fact that in health it always lies a little lower than its fellow; while the position of the heart's apex and the special resonance of the stomach will afford corroborative evidence. It has, besides, been shown by Edwin Harrison, that the exact situation of the vault of the diaphragm may, in many cases, be rapidly determined by inspection and application of the hand. The mode of proceeding varies according to the shape of the thorax, which is, with reference to this investigation, of two kinds:—1. If the width of the chest be greater just above, than precisely on, the level of a line drawn transversely across from the lower part of the ensiform cartilage,—in other words, if a slight lateral depression correspond pretty

accurately to that level—a very simple method is described by this observer for discovering the position of the upper edge of the diaphragm. Let the hand be passed from below upwards along the side of the chest, with its inner edge kept closely to the surface and the palm somewhat everted, and that inner edge will sink into a narrow sulcus situated somewhat higher up than the lateral bulge just referred to. This sulcus, which may or may not be on the same level on both sides, indicates the precise height of, and corresponds to, the vault of the diaphragm. 2. If the width of the chest be less immediately above, than on the level of, the ensiform cartilage, this rule will not apply: however, the position of the left half of the septum may then be detected by the beat of the apex of the heart; and the right half is at least not lower than its fellow. In the main these guides are correct; but exceptions occur. Thus age, by enlarging the bulk of the lung through distended atrophy, or by diminishing that bulk through simple atrophy without distension, changes the position of the wings of the diaphragm, without affecting that of Harrison's sulcus. The sulcus is impressed on the side in youth, and remains unaltered in age. So, too, I have found that, where prolonged tight-lacing had displaced the liver and diaphragm, the sulcus had, for the same reason, ceased to correspond with the upper part of the arch.

310. In *disease* the wings of the diaphragm may be both raised; both depressed; or one only may be depressed or raised, its fellow remaining *in statu quo;* or one may be raised and the other depressed; or the central tendon may be specially depressed.

311. In order to determine with precision the nature and amount of disease effecting these changes, I have been in the habit for some time of noting the position in the dead body of both wings of the diaphragm, before the chest is opened, and find in my hospital-books thirty-three cases proper for analysis in this point of view. The highest position observed in these cases was the second intercostal space,—the lowest, three

inches below the false ribs; the relative frequency with which the arches reached different heights of the thorax was as follows:*

	Right.	Left.
Second space	1	1
Third rib	–	–
Third space	8	1
Fourth rib	7	5
Fourth space	8	4
Fifth rib	6	13
Fifth space	1	7
Sixth rib	1	1
Below false ribs	1	1
	33	33

Hence it appears that in three-fourths of the cases the right vault lay *above* the fifth rib, while in two-thirds of the whole the left lay *opposite* or below it; and further, that the right wing in disease most commonly lies between the third interspace and fifth rib (in $\frac{29}{33}$ of the cases); whereas the habitual range of the left wing is from the fourth rib to the fifth interspace (in $\frac{29}{33}$ of the cases).

Next, setting aside two cases of empyema, and one of ascites, I find that the right wing lay higher than the left in twenty-one persons; the left higher than the right in two; while both were on the same level in seven. Excluding the same three cases, the amounts of difference between the heights of the two wings varied thus:—

	Right wing, above left.	Left wing, above right.
By half a rib or space	12	2
By a rib or space	5	
By a rib and a space	8	
By two ribs and a space, or two spaces and a rib	1	

Hence in nearly half the cases, where the right wing lies higher than the left, the excess of elevation only amounts to about half an inch.

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* It is to be remembered, that after death the collapse of the lungs draws the diaphragm slightly, but very slightly, upwards.

312. But what are the morbid conditions connected with these variations in the position of the diaphragm? The case of elevation to the second interspace was one of enormous ovarian and ascitic accumulation of old standing; both wings were equally raised: those of great depression (the diaphragm being highly convex downwards) were examples of profuse pleural accumulation, solid and fluid, and fluid and gaseous. These are the kinds of affection which seriously modify the position of the septum. A case where both arches lay opposite the sixth rib, was one of vesicular emphysema, uncomplicated with any other change of consequence. In the other cases a variety of conditions existed of opposing tendency; but a fair consideration of all leads to the conclusions—that rarefying diseases of the lung mechanically depress the diaphragm on one or both sides; that chronic condensing, because contracting, diseases raise it by a force of suction; that pleuritic adhesions, considered independently, have rather a depressing influence than otherwise: and lastly, that the discovery during life of any disease, even if it be highly marked, which tends *per se* to modify the position of the diaphragm, does not justify the assumption that it has so modified it in the particular case; for some counteracting, though less obvious, influence may be at play.

313. Now it follows from the last proposition, that the position of the arches of the diaphragm in disease can only be ascertained by direct observation. Harrison's sulcus, remaining as it does a fixture, tends to deceive rather than enlighten; and, though the possibility of the change, in cases of old-standing disease, is conceivable, I have never found a *new* sulcus corresponding to the altered site of the diaphragm. The vocal fremitus and the results of percussion are in truth the only real and trustworthy guides to the line of union of the chest and abdomen.

314. Extensive double emphysema, pericarditic effusion, and hypertrophy of the heart, lower the central tendon.

315. The *liver*, *spleen*, and *stomach* may likewise be raised above, or depressed below, their natural level, by conditions altering the position of the diaphragm; and thus become affected with *Elevation*, or *Procidentia*. These alterations of position are more readily detected in the case of the liver than of the other organs named; and have for this reason attracted more attention on the right than left side. Great distension of the stomach with gas will carry its amphoric percussion-note actually as high as the axilla.

SECTION VIII.—PHYSICAL CHARACTERS OF THE AIR OF EXPIRATION.

316. The variations in the physico-chemical characters of the air of expiration are of occasional diagnostic signification,—and would prove so much more frequently had they been more fully studied.

317. The temperature of expired air in health has been calculated at 99·5° Fah. by Valentin, at 98·6° by Moleschott,—the surrounding atmosphere being of medium temperature. In estimating its rises or falls in disease, the number of respirations per minute must always be taken into account; as the longer inspired air stagnates in the chest, the warmer will it make its exit: the temperature of the expired air and the frequency of breathing are, as a rule, inversely as each other.

318. The temperature sinks in pneumonia both from the diminution of surface, apt for chemical action, and the increased frequency of respiration. In various other pulmonary diseases, a similar fall in the thermometric heat of the expired air occurs through the same mechanism,—for instance in asphyxiating bronchitis. In various general diseases, implicating the blood, the expired air is more or less cool,—strikingly so in cholera Asiatica.

319. The expired air rises above the natural temperature in acute febrile diseases, during their period of sthenic reaction.

320. The quantity of watery vapour held in solution by the air of expiration varies,—from the experiments of Moleschott it appears sometimes to fall below, sometimes to reach, the saturation-point.

321. It is said that the expired air in the algide stage of cholera sometimes becomes visible, with a surrounding atmospheric temperature producing no such effect on the breath of healthy individuals. This would prove an excess of watery vapour.

322. The air and moisture of expiration in the state of health are odourless at the moment of their exit; if they be kept in closed vessels, however, putrefaction occurs, and ammonia is evolved.

323. In disease very sensible peculiarities of odour are observable: in saccharine diabetes, a sweet hay-like smell; in Bright's disease, a special modification of urinous odour; in phthisis, a faint nauseous odour, *sui generis*, is frequent, and when plainly present is actually not without diagnostic signification.

CHAPTER II.

PHYSICAL EXAMINATION OF THE HEART AND GREAT VESSELS.

INTRODUCTION—CLINICAL TOPOGRAPHY OF THE HEART AND GREAT VESSELS.

324. The heart, seated in the lower part of the anterior mediastinum, is held *in situ* by the great vessels, arterial and venous, and, through its pericardial covering, by the diaphragm. Lying obliquely, with its long axis directed forwards, downwards, and from right to left, the base of the organ corresponds, anteriorly, to the third, and the apex to the sixth, rib; posteriorly the base lies opposite the sixth and seventh dorsal vertebræ, separated from them by the aorta and œsophagus. The postero-inferior surface of the organ lies upon the central tendon of the diaphragm; the supero-anterior is in apposition partly with the lungs, partly with the walls of the chest.

325. Occupying the entire of the lower sternal, and certain portions of the left and right mammary, regions, the different parts of the organ are variously related to the surface. It is to be understood that the following topographical statement is only strictly correct, when the body is in the recumbent posture. The different landmarks were obtained by driving long needles into the chest of male subjects, at various points perpendicularly to the surface, before the abdomen or thorax had been opened.

326. The right auricle reaches more or less far, according to the state of the cardiac circulation, into the right mammary

region, on the level of the third cartilage and interspace. The right ventricle, mainly filling the lower sternal region, and coasting by its inferior, and nearly horizontal, border, the

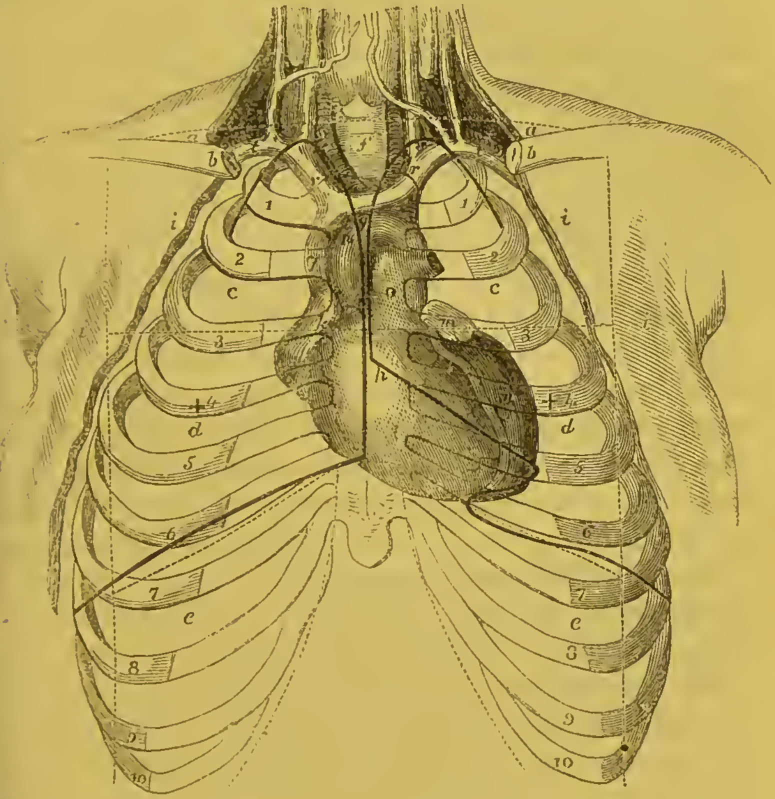

Diagram II., exhibiting the relationship of the heart and great vessels to the lungs (in moderate inspiration), and to the regions of the chest. 1 to 10 inclusive, ribs; *a*, supra-clavicular region; *b*, clavicular; *c*, infra-clavicular; *d*, mammary; *e*, infra-mammary; *f*, supra-sternal; *g*, upper sternal; *h*, lower sternal; *i*, integuments turned back; † †, nipples; *k*, right auricle; *l*, right ventricle; *m*, left auricle, appendix mainly seen; *n*, left ventricle; *o*, pulmonary artery; *p*, arch of aorta; *q*, vena cava superior; *r r*, innominate veins; *s*, innominate artery; *t t*, subclavian veins. The dotted lines indicate the outlines of the regions; the dark lines, the edges of the lungs. The heart and vessels are supposed to be full.

articulation of the sternum and ensiform cartilage, encroaches a little, at its base, on the right mammary region, about the fourth cartilage, and the two interspaces next above and below,—and besides by its apex stretches into the left mammary region, at the fifth interspace.

327. The left auricle corresponds to the third left cartilage, and a part of the interspace immediately below; the left ventricle to that portion of the left mammary region, lying a little within the vertical level of the nipple, and between the third and the fifth interspaces, or the upper border of the sixth rib.

328. The heart, then, as a whole, extends, vertically, from the third cartilage to the sixth; and transversely, from a little within the left nipple to about a finger's breadth to the right of the sternum: these are the limits of the *deep cardiac region.* The entire of the left ventricle, the greater part, by far, of the left auricle, and a fair portion of the right ventricle, towards the apex, lie to the left of the sternum; and on the level of the fourth cartilage, the widths of heart-substance, lying on either side of the left border of the sternum, are very closely the same. The most central part of the organ lies pretty accurately behind the sternal edge of the fourth left cartilage, or the upper angle of the fourth interspace. The organ corresponds posteriorly to that portion of the dorsal spine comprised between the fifth and eighth vertebræ.

329. The entire of the right auricle, and about one-third of the right ventricle, are covered by the right lung, which descends vertically downwards, coasting the middle line to the lower edge of the heart: the entire of the left auricle, the upper left part of the right ventricle, and the entire of the left ventricle, except a variable, but small, portion towards the apex, are covered by the left lung. The portion of heart uncovered by lung, thus belonging almost exclusively to the right ventricle, is of rudely triangular shape. The upper angle of the triangle corresponds to the middle line on the level of the fourth cartilage, where the anterior edge of the left lung diverges from its fellow; while the base is formed by that portion of the lower edge of the heart lying between the middle line and the spot at which the apex

beats: this is the *superficial cardiac region.* The vertical side of the triangle measures on an average, in the middle-sized adult male, two inches,—the horizontal, two inches and a half,—the oblique, nearly three inches. (Vide Diagram II.)

330. The pulmonary valves are seated opposite the junction of the edge of the third left cartilage with the sternum; the aortic a very little lower and further inwards; the tricuspid and mitral valves lie at mid-sternum (the former in front of the latter), on the level of the third interspace. The transverse distance between the right pair of valves averages half an inch: the left lie side by side; the aortic, a quarter of an inch higher than the mitral. An area of half an inch will include a portion of all four; an area of about a quarter of an inch a portion of all except the tricuspid.

331. The aorta, rising nearly opposite the union of the two sternal regions, ascends at once into the upper, at first under cover of the pulmonary artery; and, inclining to the right, reaches the inner and upper part of the second right costal cartilage: thence crossing, almost horizontally, the upper sternal region, on the level of the first interspace, and in front of the trachea just above its bifurcation, it passes backwards and downwards to the left side of the body of the third dorsal vertebra. Sometimes the entire of the arch lies a little higher than this.

332. The pulmonary artery ascends from the position of its valves, with slight inclination to the left side, as far as the second cartilage, its point of bifurcation; in this short course it passes somewhat backwards, and is consequently further somewhat from the surface of the chest opposite the second, than the third, cartilage. It occupies a portion of the upper sternal region, encroaching on the edge of the left infra-clavicular.

333. The arteria innominata rising on the level of the first interspace, behind the right half of the upper sternal region, passes upwards and to the right, bifurcating to the right of the trachea, and behind (or a little above, and to the right of) the

sterno-clavicular joint: in its course it lies in front of the right half of the trachea.

334. In as much as the sounds of the aortic arch, the pulmonary and the innominate arteries are severally best isolated at the second right, the second left, and the first right, costal cartilages,—these cartilages may for clinical purposes be respectively called the aortic, pulmonary, and innominate.

335. The superior vena cava lies along the right edge of the ascending portion of the arch of the aorta, near the right border of the sternum, from the first interspace to the third cartilage; in the former spot occurs the union of the two innominate veins.

336. The cavity of the pericardium reaches superiorly as far as the level of the second ribs.*

337. The alterations of form, size, and axis, which, vivisections prove, attend the action of the heart, are not appreciable clinically: certain accompanying changes in position can be distinguished. During the systole the organ twists spirally on its longitudinal axis from right to left, especially towards the apex, which at the same time comes forward; the converse movements attend the diastole. But other modifications of position present themselves, independently of disease. In some persons the organ lies naturally a little higher or a little lower than the average; still any variation of this kind rarely exceeds the breadth of half a rib. The habit of tight-lacing in the female tends either to raise or depress the heart, according as the line of maximum pressure is low or high; the tight abdominal belt worn by some males, to raise it slightly. Alteration of posture of the body affects the site of the organ also,—the heart falls downwards somewhat (if its substance be weighty, the fall may equal an inch) in the erect position, and comes more forward than in decumbency; changing the position in decumbency from the right to the left side will carry the heart

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* In rare instances where the sac is perfectly free from present disease, its cavity reaches the first rib; *e. g.*, case of H. Hodson, U. C. H., Males, vol. ix., p. 20. There were here three old white patches however.

an inch, or even more, to the right or left of the position it occupies when the individual lies on the back.* Inspiration, by carrying the diaphragm downwards, lowers the heart, sometimes by an entire interspace; and, by bringing a thick stratum of the left lung in front of the organ, removes it somewhat from the thoracic walls; the weakening effect on the heart's impulse and sounds thus produced is in healthy persons very perceptible: the position of the valves and the maximum points of the heart's sounds are proportionably lowered; but it is to be remembered, that the depression of the diaphragm displaces the base more than the apex. This depression of the heart tends to lengthen momentarily the great vessels, especially the ascending portion of the arch of the aorta.

SECTION I.—INSPECTION.

338. *In Health.*—Inspection directs itself to: the form of the cardiac region; the condition of its integuments; and the visible impulse of the heart and great vessels.

339. In perfectly sound chests the part of the walls lying to the left of the middle line, and corresponding to the heart, does not differ perceptibly in form from that placed to its right: these two divisions of the thorax are symmetrical. But individuals, who have never suffered from pulmonary or cardiac disease, occasionally present a moderate excess of convexity of the cardiac region, as a result of natural conformation, of curvature of the spine, or of change of form produced by influences of a non-morbid kind. So, too, physiological depression, or flattening, of the lower part of the præcordial region sometimes occurs; but M. Woillez states that such depression, when really non-morbid, is observed in the corresponding right region also, —is in fact symmetrical.

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* This is a point of considerable importance, as it shows that within certain limits, lateral moveableness of the dull sound of the deep cardiac region cannot be accepted as evidence of fluid in the pericardium.

340. The præcordial interspaces are of the same width, and lie on the same plane as their fellows on the opposite side; and the soft parts have the same characters on both.

341. In the majority of healthy persons the heart's impulse is visible only at the apex, which beats in the fifth interspace, and somewhat against the sixth rib, about midway between the line of the nipple and the left border of the sternum; the area of visible impulse does not exceed a square inch. Various physiological acts modify the precise spot of visible impulse. Thus changes of posture elevate, depress, throw it upwards or backwards: inspiration lowers it somewhat, and by carrying the lung in front of the heart weakens its force; expiration has the converse effects. A full meal or flatulent distension of the abdomen raises the apex-beat somewhat, and throws it to the left; pregnancy has the same effect. As a rule, thin, tall persons have an impulse of greater visible extent than the short and stout: in the obese, none can be detected; in persons with short sternum, it can be seen in the epigastrium. Habitually, it is more extensive in males than in females, and in persons of nervous than of other temperaments.

To the eye the impulse seems gently heaving and gliding, of brief duration, free from abruptness, regular in rhythm, and single.

342. *In Disease. Form.*—The cardiac, or præcordial, region becomes arched forwards in the course of pericardial effusion. I have never observed this change of form in pericarditis previous to the occurrence of liquid effusion; but it may occur while the fluid is yet very small in quantity. The intercostal spaces widen, and eventually almost protrude beyond the level of the ribs; while the left border of the sternum is pushed more or less, never more than slightly, forwards: change of form so marked as this indicates abundant effusion, and may reach from the sixth to the second left cartilages inclusive. Hypertrophy of the heart, especially of the left side, increases similarly the convexity of the cardiac region from the third to the seventh cartilages,

and widens, but does not produce actual bulging of, the interspaces: pericardial adhesions, especially if associated with agglutination of the pericardium to the sternum, increase the prominence caused by any given amount of co-existent hypertrophy. Solid accumulations in the lower part of the anterior mediastinum likewise arch the superjacent walls, and have occasionally caused great obscurity in diagnosis.

343. On the other hand, depression or excavation of the præcordial region may occur during the absorption-period of pericarditic effusion: commonly at the lower part of the region; occasionally, as I have once seen,[*] above its natural limits. In this instance the excavation formed during convalescence at the first and second left interspaces, close to the sternum. It has been the habit, on speculative grounds, to ascribe any such depression following pericarditis to the influence of adjacent pleurisy; but in point of fact, the change of shape sometimes occurs where all physical signs have argued against the existence of the latter inflammation.

344. *Integuments.*—The integuments of the cardiac region are sometimes markedly more œdematous than other parts of the chest, in cases of pericardial effusion lapsing into the chronic state; the phenomenon, in itself unimportant, derives interest from some auscultation-signs, which may be traced to it; these have been already described [232].

345. *Impulse.*—Disease alters the position, extent, force, character, and rhythm of the heart's visible impulse.

346. The apex-beat is in the first place changed in position by a variety of diseases of the lungs, pleura, mediastinum, and abdominal organs[†] already referred to; but affections of the

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[*] Vide case of Craddock, Clin. Lect. "Lancet," loc. cit. p. 144.

[†] Enlargement of the right, as well as of the left, lobe of the liver, may displace the heart's point upwards and outwards, and sometimes give useful and unexpected aid in diagnosis. Such displacement contributed much to distinguish acute abscess with enlargement of the liver from abscess of the abdominal walls, in a case which I published some time since. (Case of Fairbanks, Clin. Lect., "Lancet," loc. cit.

heart itself, and of its external covering likewise displace it. All enlargements of the heart depress the apex-beat, and may carry it as low as the seventh interspace, or eighth rib: if both sides of the organ be equally affected, that point is commonly displaced to the left; if the right side be the main sufferer, the impulse is chiefly visible to the right, behind and below the sternum; where the left side is alone, or chiefly, diseased, the apex may be seen at variable distances from the natural spot, to about three and a half inches to the left of the nipple.* If, as is not very uncommon, hypertrophy affect, almost exclusively, the part of the left ventricle adjoining the mitral orifice, the base of the organ falls disproportionately to the rest, and the apex is thrown forwards. Procidentia of the apex in general hypertrophy is, *cæteris paribus*, greatest, where there is agglutination of the pericardial surfaces also. Aneurism of the arch of the aorta, or of the pulmonary artery, likewise lowers mechanically the point in question. It has been affirmed that atonic relaxation of the great vessels occurs in various acute adynamic diseases to sufficient amount to allow of an appreciable fall in the heart's level: I have lately observed this in a case of typhoid fever.

347. On the other hand, diminution of the size of the cavities, consequent on sudden enormous loss of blood, acting in conjunction with retraction of the great arterial trunks, slightly raises the apex-beat. It seems possible that a rude estimate of the degree of hæmorrhage might be formed through this changed position of the heart,—but of course only in individuals the precise point of whose normal beat was previously well known.

But the affection which most notably raises the apex-beat is pericardial effusion. When considerable accumulation exists in a pericardium free from old or recent adhesions, if the heart be not enlarged, and if there be no adjacent pleuritic adhesions, the increase of fluid pushes the base of the heart and the great vessels backwards and upwards, and twists the apex outwards and upwards, so that it beats opposite the fourth interspace or rib directly behind, or a little outside, the nipple. The progress of elevation may be traced from day to day, as of the subsequent fall during the course of absorption. When the spot of the apex-beat is thus raised, it may always be *seen*, unless where general undulatory impulse tends to throw it into the shade: but it may or may not be *felt;* if not, the amount of effusion is in all probability very considerable. If during convalescence the apex gradually fall to below its natural site, hypertrophy, as an immediate sequela of the pericarditis, may be diagnosticated; for had the enlargement preceded the serous effusion, the apex would not have been raised.

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* The furthest point I remember to have seen is $3\frac{3}{8}$ inches outside the nipple (*vide* case of Hope, Clin. Lect. "Lancet," loc. cit. p. 415); this, too, is only possible in broad-chested persons.

I have recently ascertained that where the pericardial surfaces are agglutinated together by recent and abundant exudation-matter, the interstitial contraction of this will sometimes suffice, unaided by fluid in the sac, to raise the apex-beat an intercostal space.*

348. In the natural state of things the maximum of the heart's visible impulse corresponds to the apex of the organ; under peculiar circumstances that maximum may be transferred to the base. Thus in a case of highly-developed hypertrophy and dilatation of the left ventricle,† where numerous circumstances pointed to the probable existence of an aneurism of the thoracic aorta behind the base of the heart, the impulse was very notably greater about the third than the sixth interspace. As the case did not terminate fatally in the hospital, I had no means of positively ascertaining whether aneurism existed or not; but I do not believe that any affection of the heart's substance alone (except, perhaps, saccular aneurism of

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* Campion, U. C. H., Females, vol. vi., p. 39, 1852.

† F. Groove, U. C. H., Males, vol. viii., p. 13, April 25th, 1849.

the left ventricle), will thus transfer the maximum amount of visible impulse from the apex to the base. I refer here to purely systolic impulse.

349. Instead of a forward movement accompanying the systole, a sinking inwards is sometimes to be seen. In some thin-chested people, with moderate hypertrophy, while the fifth interspace rises, the fourth sinks, with the systole; the organ appears to go through a see-saw movement, and produces a momentary tendency to a vacuum at the fourth. So, too, the upper part of the epigastrium sinks in sometimes with the systole,—a sign originally deemed diagnostic of pericardial agglutination, and certainly, though more frequently absent, sometimes attending this state. I have known this systolic depression extend to the lower part of the sternum, and two or three adjacent cartilages, in hypertrophy with agglutinated pericardium. Hypertrophy with dilatation will produce epigastric depression during the systole, in persons with short sternums, independently of adhesions.

In some rare cases, the visible impulse appears horizontally double and pendulum-like.*

350. The other conditions of impulse, except the so-called undulatory variety, are better appreciated by the hand than by inspection. Undulatory impulse, suggesting the wavy motion of fluid, very variable in extent, may reach vertically from the first to the sixth interspace, and transversely from the left nipple to an inch to the right of the sternum. Its position

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* "Systolic action of heart, unnaturally visible in fifth interspace, from a finger's breadth to left of sternum to two fingers' breadth to left of nipple, or about three inches; movement distinctly pendulum-like,—a first blow appearing to be given at the outer part of space mentioned, a second at the inner; but with the finger it is found that the real impulse is only at the site of the external visible impulse. When the outer point is struck by heart's apex, the inner point sinks in; when the apex recedes, the inner point recovers its natural position." Dunn, U. C. H., Males, vol. vii., p. 76; Feb. 17th, 1852. The case was one of cancer of the stomach with great emaciation, no cardiac disease.

may be altered, in the same directions as the heart itself, by changing the patient's posture. Commonly the axis of undulation is diagonal, from below upwards, and from left to right: undulation may be well marked in one interspace, imperfect or null in an adjoining one; in this case the line of current is generally horizontal.

When so fully developed that there can be no mistake about the fact, undulatory impulse is a positive sign of fluid in the pericardium: but the sign unfortunately exists in a small proportion only of cases of hydropericarditis; and a kind of pseudo-undulation is not an uncommon character of the impulse of weak, dilated, and fatty hearts, especially where any physical conditions of the lungs or pleura, combining with its own enlargement, tend to bring an abnormal extent of the heart's surface in contact with the chest-walls.

SECTION II.—APPLICATION OF THE HAND.

351. By application of the hand, the heart's impulse, the movement of the præcordial ribs, and the state of vocal fremitus over the cardiac region, are examined.

352. *In Health.*—The visible movement corresponding to the heart's point is felt to possess a certain amount of impulsive force; which depends on the shock of the apex, and of this alone, against the side. The essential clinical fact, connected with this shock, is its synchronism with the systole of the ventricles, and the first sound of the heart; its mechanism is not thoroughly understood.*

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* It seems unnecessary in a purely clinical treatise to examine the heterodox doctrine that the healthy impulse is synchronous with the diastole of the ventricles, as more or less ingeniously argued by Burdach, Beau, Cartwright and others. And as no hypothesis of the mechanism of the impulse, yet put forward, is of direct clinical import, there is no need of considering the host of theories which have recently sprung up in Germany. The objections

Though, of course, essentially the same in position as the visible impulse, the shock may be felt a little lower than seen, namely, behind the sixth rib. Slight in amount and imperceptible to the individual himself, mixed impulsive and gliding in character, free from abruptness or sharpness, yet decisive in rhythm, of brief duration, the force of the impulse is directly as the muscularity of the heart, and the energy and rapidity with which its fibres contract. However, in broad-chested and stout persons, though provided with powerful hearts, the impulse may be scarcely perceptible to the hand : it may then be detected by placing a stethoscope in the fifth interspace, and applying the ear as if for auscultation. In thin, tall persons, the impulse is disproportionably strong,—so also in women and children, in whom, too, it is distinguished by a certain abruptness and shortness.

353. Forced expiration widens the area, and increases the apparent force of the impulse ; inspiration affects both conversely. The varying condition of the neighbouring edges of the lungs fully explains this. Various acts quickening the heart's action intensify the shock—muscular exertion, walking at a rapid pace, and *à fortiori*, running, going against the wind, ascending heights, &c., have all this effect. Voluntary acceleration of the respiration will, especially in peculiar constitutions, act (indirectly, by quickening the circulation) similarly on the impulse ; so too will the slight erethism of the circulation that accompanies digestion. But of all causes which act in this manner, independently of disease, moral influences and the passions are the most powerful ; and among these fear, perhaps, holds the highest place. The shock, under the agony of fear, becomes so powerful, as not only to be distinctly perceptible to the individual, but to be actually and sharply painful. Simple "nervousness" acts, to a slight degree, similarly.

of Valentin, Messerschmid and Kiwisch, seem fatal to the "Segner's water-wheel," or recoil, theory, ascribed to Gutbrod, and adopted by Skoda with certain qualifications.

354. *In Disease.*—In adynamic diseases, and in various non-febrile blood diseases, attended with depression of vital force, the heart's impulse habitually falls in strength.

Certain affections of the brain and spinal cord weaken the force of the heart's impulse; as do also certain medicinal substances,—aconite, digitalis, and hydrocyanic acid.

Consolidation of the adjacent edges of the lungs, or abundant solid induration-matter in the contiguous pleura, solid substances in the mediastinum, and upward enlargement of the liver or spleen, lead to exaggeration of the heart's impulse; solid material being better fitted for conducting the heart's motion than spongy lung. Diminished size of the lungs, especially of the left, and pleuritic adhesions so placed as to withdraw the edges of the lungs from in front of the heart, or to cause by pressure atrophy of the enclosed parenchyma, intensify the palpable shock for obvious reasons. The falling in of the chest-wall, which ensues on these conditions of the lung and pleura, acts in the same manner. In all these cases, it will be observed, the exaggeration in force, and extension of area are *surface-appearances*, and do not indicate any real increase of either on the part of the heart itself; many a supposed hypertrophy of the heart is nothing more than a simulation of the disease by some one of the physical conditions now referred to. On the other hand, emphysema and hypertrophy of the lungs, especially of the left, by bringing an unnatural amount of pulmonary tissue between the heart and parietes, weaken the palpable impulse, and may completely mask the direct parietal shock of well-grown cardiac hypertrophy.

Again, the quantity and quality of the circulating blood affect the heart's shock: at least the excited and sharply forcible impulse, occurring during reaction after hæmorrhage, appears in some part (perhaps frequency and force of propulsion in lieu of quantity propelled) due to the diminution of blood at command. In the spanæmia of chlorosis, the impulse, though sharp rather than strong, is yet stronger than in health. The condition of the heart's substance does not explain this.

355. The diseased states of the heart which produce *real* increase in the force and area of impulse are,—morbid functional excitement (all the varieties of purely dynamic palpitation, angina pectoris, the paroxysm of "cardiac asthma," &c.), inflammation (cardiac and peri- or endo-cardial), and enlargement, especially if combined with adhesions of the pericardium. The influence of inflammation is merely functional and dynamic; increased impulse only exists at the outset of pericarditis, before effusion has occurred to any extent. The influence of enlargement, on the other hand, is organic and statical: pure hypertrophy increases to its maximum the force of impulse; hypertrophy with dilatation, the force and area combined; dilatation weakens force, extends area.

The impulse may be increased to such a degree as to shake forcibly the head placed on the stethoscope, nay, even to shake the entire body of the patient, and the bed on which he lies; and it may stretch diagonally from the eighth left rib to the first right interspace near the sternum, and transversely, in the third and fourth interspaces, from two inches to the right of that bone to as many outside the left nipple: in such cases the impulse is very distinctly perceptible even in the left back. Between this, the maximum amount almost ever witnessed, and the natural state, all possible gradations are observable. Increased force, when extreme, is always combined with other modifications of impulse.

356. The force of impulse is lessened by effusions in the pericardium,—more readily in cases of passive than inflammatory accumulation, because in the former no excitement of the heart exists. Dilatation weakens the shock; fatty infiltration with softening has the same effect, although, on account of coëxistent enlargement, the positive amount of impulse is above the average of health; abundant deposition of fat under the cardiac pericardium generally enfeebles the impulse.

357. Increased impulse may be especially perceptible either behind the lower part of the sternum from the fourth to the seventh left cartilage, and at the epigastrium,—or below and about the left nipple, and between this and the left costal cartilages; in the former case the right ventricle is commonly most affected, in the latter the left. But exceptions to this rule occasionally occur; extreme relative hypertrophy of the right ventricle will throw the impulse so much to the left of the sternum, as to simulate that of enlargement of the left side of the organ.*

358. If increased impulse be mainly traceable in a horizontal direction, the right ventricle is by some held to be the source of the increase, because enlargement on that side has a tendency to render the position of the heart more completely horizontal; whereas if mainly traceable in a vertical, or vertico-diagonal direction, the left ventricle is at fault, inasmuch as its hypertrophy elongates the organ in those directions. I have occasionally found the former, frequently the latter, of these propositions correct; but neither is worthy of implicit clinical trust. Hypertrophy of the left ventricle, when extensive, widens the impulse horizontally.

359. The character of the impulse varies as much as its force. The *apex-beat* may retain the natural impulsive and gliding character, and be merely increased in power. Or it may acquire a quick, abrupt sharpness; or convey the sensation of slow heaving, or pushing forwards against an obstacle: in hypertrophy of the left ventricle, the latter character is habitual; in dilatation the former. Where weakened, the loss of power may be simple; or the shock be also abrupt and jerking, or marked by fluttering unsteadiness, as in various species of softening. The *general impulse* on either side of the heart may vary similarly.

360. In rhythm, the heart's natural impulse, we have seen,

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* Kernis, ætat. 10, U. C. H., Females, vol. ii. p. 237, 1847.

is synchronous with the systole of the ventricles, and single. Now the impulse lags slightly behind the systolic sound of the heart, where fluid is accumulated in the pericardium, and the position of the apex is *not* elevated; before the impulse reaches the chest-wall, a stratum of fluid must be pushed aside. Besides in certain states of disease, the shock becomes double, the added impulse being systolic or diastolic. Double *systolic* impulse is an occasional, though rare, attendant on eccentric hypertrophy, a not uncommon one (it may even be treble, whence a peculiar jogging, shaking character) where, in addition to the hypertrophy, the pericardial surfaces are agglutinated, or covered with recent exudation-matter.* Laennec noticed that in cases of great hypertrophy it seems " as though the heart swelled, and applied itself to the parietes at first by a single point, then by its whole surface, and finally sank back in a sudden manner." The sudden " sinking back" of the heart, Hope was the first to notice, sometimes terminates in a jog or shock (back-stroke) obviously *diastolic* in time. He believed this " diastolic impulse" was strongest in dilated, though sometimes very considerable in simple, hypertrophy,—and imperceptible in simple dilatation: this accords with my experience also. The explanation of the phenomenon given by Hope, seems, however, scarcely intelligible. " It is occasioned by the diastole of the ventricles, during which action the heart sinks back from the walls of the chest, and this sinking back terminates in a jog or shock, *occasioned by the refilling of the ventricles*, and constituting the diastolic impulse in question." But the mere expansion of dilated hypertrophous ventricles is doubtless in itself capable of giving a strong blow; the intense power of the natural diastolic expansion has always been noticed by vivisectors. On the other hand, there are many cases in which the diastolic sensation is rather one of *inward succussion* than *parietal impulsion;* it then depends, quite as probably, on falling back of the weighty heart against the spine,—a view strengthened by the fact that "diastolic impulse" is peculiarly obvious in cases where solid accumulations, aneurismal or other, lie behind an enlarged heart.* A *double* diastolic impulse has been described; this I have not observed,—it appears not improbable that impulses, so spoken of on the ground of their being unattended with arterial pulse, were in reality systolic, but too weak to reach the radial arteries.

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* Courtenay, U. C. H., Females, vol. vii., p. 303., et seq.

361. In the normal state, any successive number of impulses are equidistant, and of precisely identical, or very nearly identical, force; in diseased conditions of the organ this uniformity may disappear. There may be several strong shocks succeeded by others as feeble; and the number of each kind may be uniformly, or very nearly so, the same,—or their number may vary greatly,—in point of time all forms of irregularity are observable: force is rarely thus affected without time being implicated also. Simple hypertrophy of one or both ventricles does not produce this perversion of rhythm and force, unless some condition seriously affecting the pulmonary circulation occurs,—especially congestions, and inflammations of the lung. But both force and time are affected to a high degree, when dilated or simple hypertrophy of the left ventricle coëxists with mitral insufficiency; force more than time, where the same state of the ventricle attends aortic insufficiency. Marked dilatation, fatty infiltration of the heart, and flabby softness, produce great irregularities of rhythm and force of impulse,—but in these cases no single impulse is, absolutely speaking, powerful, unless the walls of the ventricles be thickened also. Pericarditis, both before and after effusion, is occasionally attended with similar irregularity; so, too, are various malformations of the heart,

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* When a heart either quite healthy, or affected with enlargement so slight as to be practically insignificant, is excited by exercise or emotion, a slight jog synchronous with the diastole may often be felt. But this jog cannot, as it appears to me, be correctly spoken of, with Dr. Bellingham, as an impulse: the impulsive character seems to be wanting.

especially during paroxysms of dyspnœa; here perversion of the relative capacities of the two sides of the heart probably acts as the immediate cause of the irregularity.

362. The impulses, whether natural or morbid, hitherto spoken of, are caused by ventricular action: does the contraction of the auricles produce visible or palpable impulse? Certainly not, in the state of health: their systole is feeble, and tends to withdraw them from the surface; their diastole yet feebler, passive and gradual in character. Where, however, the auricles are hypertrophous and dilated, it is conceivable that their systole, perhaps even their diastole, may become perceptibly impulsive. Auricular impulse, if systolic, should very slightly precede the shock of the heart's apex; if diastolic, it might coincide with any moment of the ventricular systole, or diastole, and probably would coincide with the commencement of the former.

In a case where percussion-dulness at the second left interspace indicated an enlarged left auricle, impulse, preceding the ventricular systole, existed in the third left interspace close to the sternum; this was probably auricular and systolic. The left ventricle was hypertrophous, and the mitral valve insufficient.* It is probable, too, as suggested by Dr. Blackiston,† that the impulse of the ventricles may be communicated, in some cases of dilatation of the appendix of the left auricle, to the second left interspace: such *pseudo-auricular* impulse would synchronise with the ventricular systole.

363. The præcordial intercostal spaces are, in the natural state, of the same form and dimensions as on the other side of the chest. They are widened by hypertrophy,—widened, and even bulged outwards, by pericardial effusion, especially in the young; narrowed by chronic pleurisy and pericarditis, particularly if there be no hypertrophy sequential to the latter.

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* Frederick Smith, U. C. H., Males, vol. v., p. 296, Oct. 1850.

† Diseases of the Chest, p. 124.

The state of movement in these spaces has been considered in the previous chapter.

364. The normal effect of the heart on the vocal fremitus of the chest has already [44] been explained. Enlargements of the organ, especially of its right division, and accumulations of fluid in the pericardium will annul the fremitus to an unnatural extent to the right of the sternum: but this is a sign of scarcely any value in practice, except where suspicion may be entertained that dulness under percussion, apparently connected with the heart, is in reality dependent on induration of the edge of the right lung.

365. Application of the hand detects, under special circumstances, two kinds of phenomena which are purely adventitious —valvular thrill and pericardial friction-fremitus.

Valvular thrill, or "purring tremor," resembles, in many cases, the vibration of the surface of a purring cat,—in others is more like the vocal fremitus felt over the larynx of persons with powerful bass voices. Varying in force in different cases and in different conditions of disease, it may change from moment to moment in the same person, according to the energy of the circulation,—mental or bodily excitement rendering it powerful, when, in the state of repose, it had been almost imperceptible. Synchronous, and agreeing in rhythm, with the ventricular systole, it is felt mainly below and within the nipple, about the fourth interspace, over an area of from one to three inches; or at mid-sternum on the level of the third rib, in the third left, and the second right interspaces close to that bone: if perceptible higher than these limits, and especially if so above the clavicle, this species of thrill ceases to be of purely cardiac origin. Habitually caused by forcible and rapid propulsion, in a rippling current, of blood—more particularly of blood altered in certain of its properties—through orifices narrowed and roughened by disease, it may in rare cases occur independently of textural change in the valves. When connected with valvular disease, it ceases, for obvious reasons, to be produced, unless a certain quantity of blood is propelled with a certain force through the diseased orifice; and hence, after existing to a high degree, it may disappear, because the narrowing which originally led to it has increased to an extreme amount, while the heart's force has, from some independent cause, more or less failed; a similar alteration, it is well known, occurs previously to death in certain organic murmurs of the heart.

The two combinations of disease, in which cardiac thrill is observed to the maximum degree, are insufficiency of the mitral valve with dilated hypertrophy of the left ventricle,—and constriction of the aortic orifice, coupled with hypertrophy of the same kind and seat: in the former case, the seat of the phenomenon is below and within the nipple; in the latter, at the aortic base. Now, the physical conditions of the phenomenon seem pretty fairly balanced in point of completeness in these two combinations; yet certainly cardiac thrill is more frequently met with as a dependance on mitral regurgitant than aortic constrictive disease. Hope supposed that this depended on aortic thrill being masked by the interposed sternum; but, although it is true, thrill, thus localised, may be sometimes rendered more distinct by causing the patient to lie on the left side, I believe the real cause of the comparative rarity to be no other than the less frequency of marked aortic than of mitral disease. I do not remember ever to have observed cardiac thrill synchronous with the ventricular diastole; tricuspid and mitral constriction,* as well as aortic regurgitation, have consequently not in my experience led to its production. The force of the current is, perhaps, insufficient in the three cases: yet, I can well conceive that in a spanæmic person, with highly developed aortic regurgitation, a minor degree of the phenomenon might exist; a loose vibratile portion of valve or vegetation, floating in the current, might also greatly facilitate its production. Neither have I ever found acute endocarditis, where there was full certainty of the absence of old-standing valvular disease, attended with thrill. But I have occasionally known a faint systolic aortic thrill, never a mitral one, accompany palpitation in spanæmic women, and in persons who had suddenly lost a large quantity of blood. I do not believe that mere nervous excitement of the heart will produce thrill in people whose blood is healthy.

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* If mitral constriction be present, the thrill of coexistent regurgitation will be thereby rendered more intense.

366. Pericardial tactile friction-fremitus, like the analogous phenomenon of pleural fremitus, is considerably rarer, and, when occurring, of shorter duration, than audible friction-sound. It has much more of a rubbing character than valvular thrill, with which it may co-exist, and is more movable than this,—migrates from one part to another of the cardiac region within the course of a few hours, although the patient has retained and is examined in the same posture. In doubtful cases it may be distinguished from pleural fremitus by causing the patient to suspend his breath, for its rhythm is of course cardiac;* an excess of fremitus commonly attends the ventricular systole. Cardiac action may be supposed capable of producing pleural tactile fremitus; but I do not remember ever to have observed it. A remarkable case has been recorded by Dr. Swett,† where a distinct thrill over the heart was caused by friction against an enlarged and "tuberculated" left lobe of the liver; the heart, as likewise the pericardium, proved "perfectly normal in all its parts." Cardiac action may then produce tactile fremitus beyond the pericardium, and even through the diaphragm.

SECTION III.—MENSURATION.

367. Mensuration of the surface corresponding to the heart, confirms inferences, otherwise deduced, as to the existence of

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* Such difficulty must, however, be rare: pleuritic fremitus produces the sensation of successive *rubs;* pericardial, that of *thrill.*

† New York Journ. of Med., July 1840, p. 6.

certain affections of the organ; and sometimes becomes a useful auxiliary in diagnosis.

368. Thus, in health, the nipples are equidistant from the middle line; in enlargement of the heart and in pericardial effusion, the distance between the left nipple and that line undergoes increase,—to a very notable amount in some old-standing cases of dilated hypertrophy. The left nipple, too, is carried somewhat downwards by the same affections. Again, the space comprised between the upper border of the third, and lower border of the sixth ribs, an inch outside the sternum, is the same on both sides in healthy persons free from curvature of the spine: the diseases just named increase the measurement on the left side. The influence of the two affections is the same on these measures; it differs in regard of the following one. The vertical distance between the left clavicle and the heart's apex-beat is in health the same as between the right clavicle, and the point on the right side, lying on the same horizontal level as, and corresponding to, that beat: now, in pericardial effusion, the distance on the left side falls considerably below, in enlargement of the heart rises considerably above, that on the right side. This admeasurement is really useful in some cases of difficulty,—as, for instance, when an enlarged and dilated feeble heart beats with a quasi-undulatory impulse.

369. In forced inspiration, the expansion on the level of the sixth cartilage, is in health slightly less on the left side than the right, a deficiency referrible to the influence of the heart. In pericardial effusion this deficiency, instead of, as might on first thought be supposed, increasing, actually disappears; the pressure of the fluid on the diaphragm throws an extra amount of work on the ribs, which comparatively move more than in health. When the fluid accumulates to a great amount, however, say twenty ounces, the præcordial ribs, also, move less than in health. I do not know how this matter stands previous to the occurrence of effusion in pericarditis; nor have I sufficiently examined the point to say positively how the costal

motion varies in the different varieties of enlargement of the heart, with and without valvular disease.

SECTION IV.—PERCUSSION.

370. *In Health.*—Numerous difficulties stand in the way of accurate discrimination of the heart's outline by percussion: above the organ lie blood-vessels of large calibre, forming a quasi-solid mass; the lungs encroach on its edges; the liver, of a density and resonance scarcely differing from its own, coasts its lower border; while in front are placed the sternum, costal cartilages and ribs, possessed of a special resonance. Add to all this, that the heart is in a state of perpetual restlessness, and constantly changing its precise bulk and form, while the quantity of lung in front of it on either side is likewise undergoing the changes dependent on respiration. Yet, in despite of all this, it is possible by care to establish, with sufficient accuracy for all practical purposes, the position and bulk of the organ.

371. The heart cannot be percussed with success except in the recumbent posture; and, unless for the special purpose of ascertaining to what extent the organ (or the source of præcordial dulness, whatever it be) is movable, not the slightest movement, even of the limbs, ought to be permitted during the examination. The force used in striking will vary from the gentlest touch with the flat surface of the pulp of the middle finger, to a sharp tap with the ends of one or more fingers; it is a mistake to suppose that violent blows serve any useful purpose.

372. Percussion of the chest, where the heart lies immediately beneath the surface, gives a short dull sound; the parietal resistance is highly marked. These characters are modified somewhat, except in the intercostal spaces, by the clearer and more prolonged resonance of the sternum, ribs and cartilages. Where the organ is invested in front by lung, the

resonance partakes of course, more or less, of pulmonary quality, according to the thickness of the intervening stratum of pulmonary tissue. We may then clinically speak of the heart's *superficial* and *deep-seated* dulness.

373. By reference to Diagram II., it will be seen that the limits of the heart's *superficial dulness* must be as follows:—on the right a vertical line, extending at mid-sternum from the level of the fourth rib to that of the sixth; on the left an oblique line passing outwards and downwards at a more or less acute angle from the latter, opposite the fourth cartilage, and curving inwards again, somewhat within the site of the nipple, to the sixth rib, beside the heart's apex; inferiorly, a line gently sloping to the left, from the central point of the lower edge of the sternum, along the sixth cartilage. This is the extent of heart uncovered by lung in calm respiration; and the form of the part is obviously, though only rudely, triangular: the lengths of its sides have already been stated [331]. The area of the triangle is to be made out only by the gentlest taps with the pulp of a finger, either on the surface directly, or on a finger of the other hand. The right border is with difficulty established, on account of the sternal resonance. Forced inspiration diminishes the extent of this area; expiration increases it, especially in an upward direction and to the right; and by the kind of percussion described above, these changes in extent can without much difficulty be ascertained. They are, however, more frequently serviceable as tests of the freedom of play of the edges of the lungs, than of the condition of the heart.

374. Beyond the limits of superficial dulness, the outline of the heart may be ascertained by the deep-seated dulness elicited by firm percussion. This *deep-seated dulness* extends normally in a vertical direction, from the third to the edge of the sixth cartilage, and transversely from the left nipple to a little beyond the right edge of the sternum opposite the fourth cartilage; the longest measurement is the diagonal one, from

the upper part of the third right cartilage (the right auricle) to the point of the apex-beat. In ascertaining these limits there are two main sources of perplexity. The first consists in the difficulty of defining exactly the line of the base; as the dulness, produced by the mass of large vessels there, is nearly as great as that of the heart itself; this perplexity fortunately only occurs, where the edges of the lungs are unusually far apart: the second, the difficulty of separating the adjacent edges of the heart and liver, has already been discussed [109]. The "auscultatory percussion" of Drs. Cammann and Clark* most certainly renders the change of pitch, on passing from the liver to the heart, more positive and definite, than it appears under ordinary percussion, and may be called to our aid in puzzling cases. The method referred to consists in receiving percussion-sounds directly through a solid stethoscope to which the ear is applied, instead of, as in the ordinary way, receiving them weakened by diffusion through the air. By practice the process becomes manageable without the assistance of a second person; the observer, having placed his ear to the stethoscope over the lower part of the liver, percusses the surface diagonally upwards and to the left, in the direction of the heart, and is apprised of his reaching that organ, by an abrupt change in the pitch of the sound. The interesting paper of the American physicians is well worthy of study.

375. Certain physiological conditions modify the exact area of the heart's dulness. Its outline cannot be identical during the different periods of the heart's revolution: the chances that percussion gives the outline in the state of ventricular systole, diastole, or of quiescence, may be represented respectively by the numbers 2, 1 and 2; the chief interest of this matter turns, on the testing the correctness of results, obtained during life, by the actual size of the organ after death; it may be neglected in ordinary practice.† Again, the act of respiration, and alterations of

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* New York Journ. of Med., July 1840.

† The heart is drawn up after death, both by the emptying of its cavities

posture of the trunk will of course change the position of the heart's dulness. The area of superficial dulness is relatively less in infancy, greater in old age, unless the lungs become emphysematous, than in manhood.

376. *In Disease.*—Various morbid conditions, independent of disease of the heart or its membranes, may increase the area of præcordial dulness. Among these may be enumerated pure atrophy of either lung, with diminished volume; consolidations of the portions of lung adjoining the heart; enlargement aud elevation of either lobe of the liver; accumulations in the mediastinum or pleura; diminution of bulk of the left lung with lateral pleural adhesions, a state tending to bring an undue quantity of the heart into contact with the chest-wall; aneurism of the great vessels; and, even, in infinitely rare cases, tumor, with constriction and pouching, of the œsophagus. In all these cases, the actual physical state of the heart may be completely unaffected. Not so, when the extent of præcordial dulness is increased in consequence of the stagnation of blood in the right cardiac cavities, caused by obstruction of the pulmonary circulation in various forms of dyspnœa; here the heart's own dimensions are temporarily changed.

377. There is only one thoracic affection independent of the heart itself which seriously diminishes the extent of præcordial dulness,—emphysema of the lungs,—particularly of the left, and especially when conjoined with bronchitis. The temporary influence of bronchitis in increasing the bulk of an emphysematous lung, and so masking the dulness of a very greatly enlarged heart, is well shown in the case already referred to.* The same case illustrates the influence of ascites in diminishing the extent of the heart's dulness, by pushing the organ upwards under cover of the lung.

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and by the expiratory collapse of the lungs: hence another of several reasons why slight differences may arise in clinical and post-mortem examinations. The mode of death must seriously influence this.

* Case of Hope, Clin. Lect., Lancet, loc. cit. p. 443.

Venesection, as first shown by M. Piorry, will very sensibly diminish præcordial dulness, especially towards the right side, in persons whose right cavities had previously been loaded with blood. A marked state of anæmia, by reducing the heart's distension, narrows the area of its dulness.

378. The area of præcordial dulness may be diminished or increased by disease of the heart itself. Diminution of that area attends primary concentric atrophy of the organ; but though decrease in bulk of the heart, and in calibre of the large vessels, often occurs to a very notable amount in the tuberculous and cancerous cachexiæ, it is seldom detected during life in either: adjacent pulmonary dulness renders its discovery difficult in the former case; it is seldom sought for in the latter, but may sometimes be discovered.

In pneumo-pericardium the natural dulness disappears more or less completely in proportion to the quantity of gas accumulated; even if there be fluid, as well as gas, in the serous sac, the entire præcordial region may give more or less tympanitic resonance in dorsal recumbency. By changing the patient's posture from the back to the side, dull sound will be elicited in the then inferior, tympanitic sound in the then superior, part of the præcordial region: this interesting fact I succeeded in establishing in a singular case of traumatic communication between the œsophagus and pericardium.*

379. The cardiac affections which widen the area of præcordial dulness, are materially more important than those just reviewed; they are referrible to three main heads: enlargement of the heart, fluid accumulation, and solid formations.

380. Hypertrophy, in all its observed forms and sites, increases the extent of cardiac dulness: the position of the increase and the elasticity of the walls differ under different

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* Case of Ramo Samee, U. C. H., under the late Dr. A. T. Thompson. The perforation was produced in the attempt to swallow a long blunt instrument, a juggler's "knife;" the case terminated fatally, and the preparation is in U. C. Museum. No. 3859.

circumstances. Simple hypertrophy of all the cavities, or of both ventricles, or the same state combined with uniform dilatation, extends the outline of dulness downwards, and to both sides, more, however, to the left than the right: scarcely any impression is produced on the upper outline of the heart's resonance,—a proposition which applies, with scarcely an exception, to all varieties of true cardiac enlargement; for, as already mentioned, the tendency of increase of bulk is to carry the heart downwards. In general dilatation, simple, or combined with attenuation, extension of dulness is also observed; and very careful percussion will detect less parietal resistance in this than in the preceding cases. If enlargement be limited to either ventricle, the extension of the dulness takes place in the direction of the affected one; and what has been said concerning the site of impulse in such cases, applies to that of dulness. Accumulation of fat under the pericardium, when sufficiently great to alter the limits of dulness, generally does so to the right side; for the simple reason that such accumulation begins with, and attains its maximum at, that side. Hypertrophy of the left auricle carries deficiency of resonance into the second left interspace.

If with the enlargement co-exist pericardial adhesions, the extent of dulness is always proportionately increased, but more so to the left than the right side: this effect is still more perceptible if there be pleuritic agglutination in the left inframammary region. In some cases of old pericarditis the area of the heart's dulness is increased upwards: thus I have repeatedly found, in persons who had previously been under my care for rheumatic pericarditis, that more or less marked dulness existed, years after, in the second left interspace, even up to the second cartilage, and at the adjacent part of the sternum. This state of resonance may exist with or without obvious enlargement of the heart; if without such enlargement, it can only be explained by the presence of solid induration-matter about the great vessels and base of the heart; if with such enlargement, it is explicable by the elevation, which the heart undergoes during the effusion-period of pericarditis, being maintained by agglutination of the pericardium, in spite of the depressing influence of the enlargement.

381. Fluid accumulation in the heart's right cavities (of blood, of course), occurs to sufficient amount, under certain circumstances of obstructed cardiac circulation, to extend very perceptibly the area of dulness on the right of the sternum. The most important condition of the kind, practically, is dilatation of the right auricle and ventricle, combined with insufficiency of the tricuspid valve. The extension of dulness takes place mainly between the second and the fifth interspaces. The accumulation of fluid and semi-solid blood in endocarditis sometimes considerably widens the area, both to the right and left.

Fluid accumulation in the pericardium, whether passively or actively dropsical, or the result of pericarditis, produces an extension of cardiac dulness, even when of small amount. I know that four ounces will widen the area of dulness,—perhaps even less than this will suffice. Clinical experience proves (artificial distension of the pericardium obviously could not be accepted as conclusive evidence) that the pericardium undergoes distension most readily upwards, with greatest difficulty downwards, with medium facility forwards, backwards, and sidewards. When distended with fluid, the sac retains its original pyramidal form,—the base below, the apex above. The level of that base at the front of the chest commonly corresponds to the lower border of the sixth rib, sometimes to the sixth interspace, in very rare instances to the seventh rib: in the latter class of cases the texture of the pericardium had probably undergone some chronic change, of a rarifying kind, before the occurrence of effusion—or at least been affected with more than average facility by fluid pressure. In cases of extreme accumulation the diaphragm is arched downwards by the fluid; the epigastrium may thus be rendered somewhat prominent, but the dulness of the fluid is with difficulty distinguishable from that of the liver,—it does not reach sufficiently to the left side to modify the percussion in the hypochondrium. The apex of the pyramid, as the fluid increases, gradually rises to the second left cartilage, to the first, to the sterno-clavicular joint, and even to nearly an inch above the clavicle, displacing the apex of the lung at the inner aspect of the supra-clavicular region. As the fluid increases, it pushes aside the edges of the lungs, where they join in inspiration at mid-sternum, between the second and fourth ribs;* and this detrusion, in cases of abundant effusion, condenses the adjacent edges of the lungs, and so increases the lateral extent of dulness: on the right side the state of vocal fremitus will sometimes guide to the line, where the condensed tissue and the fluid join. With equal superficial extent the dulness from pericardial fluid is more absolute, and the parietal resistance greater, than from hypertrophy;—this probably depends on the more perfect approximation of the fluid, than of the solid, material to the walls. This distinction is too delicate to be trusted to at the bed-side; the dulness of effusion is better distinguished from that of general hypertrophy by its extensive range above the third rib, and its ordinary limitation to the sixth rib below; and from dilated hypertrophy of the right or left ventricle by not extending, disproportionately, to the area of dulness, towards the right or left side. It must not be forgotten that an aneurismal sac, (we may suppose it filled with fibrine, pulseless, latent, and *pro tanto* deceptive,) of the transverse part of the arch of the aorta, and bulging inferiorly; or a small mediastinal tumor; or even superabundance of natural fat, placed just above the third left cartilage, and behind the sternum (a source of fallacy more frequent in persons with much subcutaneous fat than in the thin); may all give to the dulness of an enlarged heart the pyramidal form of that dependent on effusion. And if an enlarged heart, with such an accidental appendage above it, be weak and flabby, and give a quasi-undulatory impulse, the distinction of the case from one of pericardial effusion, especially if the commemorative history be imperfect, becomes one of most serious difficulty.

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* Bartlett, U. C. H., Males, vol. iv. p. 292. Such extraordinary distension could only result from very slow progress of the disease: it appeared to have lasted in this case six weeks. The lungs were *five* inches apart on the level of the second rib.

382. The plastic exudation-matter of pericarditis sometimes forms a layer, one-third to three-fourths of an inch thick, of solid substance applied to the heart's surface : if chance place this great thickness of substance on the lateral confines of the organ, the area of dulness must be proportionately increased; but the sign is one of too great delicacy to be clinically serviceable.

Cancerous accumulation in, or underneath, the pericardium affects the præcordial resonance in proportion to its amount.

383. The *quality* of the percussion-sound undergoes no very material change under the circumstances hitherto referred to, except in cases of pneumo-pericardium ; but in those very rare morbid states, induration of the heart's substance or pseudo-ossification of the pericardium, the sound must become more ringing and osteal in quality than natural,—at the same time the resistance of the parietes would be sharp and highly marked.

384. The *form* of *deep-seated* dulness, peculiarly affected, as we have seen, by pericardial effusion, remains essentially unchanged by general hypertrophy ; excessive hypertrophy of any particular part must of necessity modify the character of its outline,—but rarely to an amount that can be trusted to at the bed-side. Dilatation renders it more or less markedly square. Again, hypertrophy alters the form of the *superficial* dulness by pushing aside the lungs, and converts the triangular, into an irregularly square, space. Solid masses under the pericardium change the outline of dulness according to the direction of their growth ; but their influence is very slight in this way, and on their rarity it is needless to insist.

385. Valvular diseases exercise no direct influence on præ-

cordial resonance: the increase of dulness so frequently co-existing with them of course really depends on some form of attendant enlargement of the heart. The membranous inflammations, when acute, though probably slightly increasing the heart's bulk by congesting its substance, do not *per se* (endocarditis at any period, pericarditis until exudation has occurred,) appreciably alter præcordial dulness.

386. Præcordial dulness, whether dependent on fluid in the pericardium or enlargement of the heart, moves from side to side somewhat, as the posture of the patient is changed. Old pericardial adhesions, even, will not prevent sideward locomotion of a hypertrophous heart,—nor will pleuritic adhesions, in front of the heart in addition, do so, unless they be close and extensive.

SECTION V.—AUSCULTATION.

387. Auscultation, directed to the heart, analyses:—(A.) Certain sounds produced by the normal action of the organ; (B.) Modifications of these occurring independently of heart-disease; (C.) Morbid states dependent on heart-disease. Besides (D.) the state of the respiratory murmurs, and (E.) of vocal resonance, specially in the præcordial region, sometimes affords useful information.

388. While the heart is ausculted, the patient should be placed in the recumbent posture, with the head slightly raised; unless, from the nature of his disease, this posture be an uncomfortable one. As much of the precision of the notions, obtained from the examination, depends on our being able to connect the spots, where various sounds are heard, with certain parts of the heart itself or great vessels, it is obvious that, in order to simplify our task, we should always auscult patients in the posture in which clinical practice must present them most frequently to us.

389. If there be any doubt about the superiority of mediate

or immediate auscultation, in the case of the lungs, there is none in that of the heart. Certain phenomena, well audible at a given point with the stethoscope, may cease to be perceptible *one-third of an inch* beyond that point: such limitation as this evidently could not be effected by direct application of the ear to the surface.

390. By forcible pressure of the stethoscope friction-sounds are commonly increased in loudness; when doubt is entertained about their existence, the doubt may sometimes be thus removed.* Sound produced by the heart's shock is similarly increased, and *pro tanto* the other elements of cardiac sound are thrown into the shade.

391. The heart's sounds are more or less masked by certain morbid states of the respiratory sounds,—and they may be given unnatural characters by the chance coincidence of the sound of natural inspiration or expiration; hence the necessity of causing the patient to hold his breath from time to time, while the heart is auscultated.

NORMAL CARDIAC SOUNDS.

392. Each complete revolution of the heart is accompanied by two successive sounds, audible in the præcordial region, and separated from each other by intervals of silence. These two sounds differ in all their characters; and the two periods of silence differ in the only character they can differ in, namely, duration: these differences are found, provided the position of the stethoscope be unchanged, to be maintained either without the smallest, or with very slight, variation in successive beats of the same heart.

393. The first of these two sounds, coincident with the systole of the ventricles, the heart's shock against the side, and the pulse, or diastole of the arteries nearly adjoining the heart,

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* Pressure, carried beyond a certain point, sometimes deadens friction sound, doubtless by mechanically obstructing the freedom of attrition.

is called the first, or systolic, or (because of maximum force at the lower part of the cardiac region) inferior, sound of the heart. The second of the two sounds, synchronous with the diastole of the ventricles, the recedence of the heart from the side, and the pulseless state, or systole, of the large arteries, is known as the second, diastolic, or (because of maximum loudness at the upper part of the cardiac region) superior, sound of the heart. The noiseless period succeeding to the first sound may be called the first, or post-systolic silence; that succeeding the second sound, the second, or post-diastolic silence.

394. If the period of an entire revolution of the heart, that is from the commencement of one first sound to the commencement of the next succeeding first sound, be divided into ten equal parts, about four of these will be found to be occupied by the first sound, one by the post-systolic silence, two by the second sound, and three by the post-diastolic silence. This estimate is, however, only to be taken as approximately true. When the pulse beats as much as from eighty to ninety times in the minute, the post-systolic silence is difficult enough of detection; but it becomes obvious, where the pulse does not exceed sixty in a minute. The estimate, also, refers only, the student will remember, to the periods of sound and silence, not to those of action and inaction, of the heart: clinically there is no trustworthy plan of measuring the length of the systole and diastole.

395. An accurate idea of the characters of the heart's sounds cannot be given by a single description of them, as heard in any one particular spot: they vary materially at different parts of the cardiac region, both in their positive and relative properties. The least study of the healthy chest will convince the student that the description which it is the habit to apply to the sounds of the heart generally, holds good only when these are heard towards the left apex. The sounds require comparative analysis: (*a*) at both sides of the apex-region, and at both sides of the base-region; (*b*) at base and apex on the same

sides of the organ; (*c*) at base and apex on opposite sides. Now this is the most difficult of all studies connected with the healthy heart; but it is essential as the basis of observation of the organ in a state of disease.

396. (*a*) At the left apex the first sound is dull, measured, booming, prolonged, and strongly accentuated; its commencement pretty sharply defined, its close much less so. Double the length of the second sound, of lower pitch than this, and seeming to the ear deep-seated, it attains its maximum at this apex, in regard of accentuation, prolongation, and measured, booming character, but not habitually in amount of sonorousness. The second sound, only half as long as the first—clear, abrupt, flapping, and short—is more sonorous, more superficial, and of higher pitch than its predecessor. At the right apex-region, the first sound is considerably clearer, shorter, more abrupt, less strongly accentuated, and of somewhat higher pitch than at the left apex-region. This difference of character depends, probably, both on the thinness of the walls of the right, as compared with the left ventricle, and on the parietes being of different conducting power in the two situations: at all events the sound, audible at the end of the sternum, may be fairly referred more particularly to the right ventricle; that near the nipple, to the left ventricle. So, too, the second sound is habitually clearer, and sometimes even of higher pitch, at the end of the sternum, than towards the left apex; this is, however, less constantly true, and I have known the converse to be the case, where no suspicion existed of the existence of dilatation of the left ventricle—where, indeed, the first sound possessed to the full its natural share of dull, prolonged, booming character.

Passing from the apex to the base, the same kind of dissimilitude, laterally, in the sounds, may be detected,—not indeed precisely at the base, but a little above this, opposite the second interspace, where the aorta and pulmonary artery are in contact. The first sound at the right second interspace is commonly duller, of slightly lower pitch, and more prolonged than at the left corresponding point: in neither is it accentuated. The second sounds differ here in the same characters and in the same manner, but to a less degree: they are accentuated in both places, more on the right side than the left.

397. (*b*) Let us now examine the two sounds comparatively at base and apex on the same sides of the heart. The first sound, strongly accentuated at the left apex,—prolonged, booming, and dull,—at the left base loses the accent, which passes to the second sound, while this becomes louder, more ringing, and sometimes even of higher pitch than at that apex. At apex and base on the right side, the characters of the first sound are very similar; it has more accent in the former than in the latter spot, however, both positively, and *à fortiori* as compared with the second sound.

398. (*c*) The relative characters of the two sounds at base and apex at opposite sides of the heart (crucially taken, as it were,) may easily be deduced from the foregoing account.

399. Now, if these statements be correct, it appears obvious that no single articulate symbol can be devised, applicable to the heart's sounds in more than one point. The subjoined series is offered for the four points of prominent clinical interest. The acute accent-mark is used to show where the accent falls,—twice, when it falls very strongly; the marks of long and short, where length or shortness of sound is a prominent characteristic.

	First sound.	Second sound.
At the left apex	ō̋ubb	dŭp
At the right apex	úp	tŭp
At the left base *	up	túp
At the right base *	ub	tűpp

The eye gathers from these symbols the tendency to equalisation in length observable in the sounds at the bases, as well as the transference of the accent from the first to the second sound at the apices and bases respectively.

400. The extent to which the heart's sounds are audible in

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* The left and right second interspaces close to the edge of the sternum.

health, is not subject to any fixed rule.* One great mistake, commonly committed by authors who attempt to define it, is not considering separately the first and second sounds. From this omission, the ordinary starting proposition, that "the heart's sounds are heard at their maximum in the præcordial region," becomes an error: the second sound is, in truth, heard in nine people out of ten, more clearly at mid-sternum, on the level of the second interspace, than at any point of the præcordial region,—even limiting that region to the space in which the heart is uncovered by the lung during tranquil breathing. The thickness of the soft parts, the form of the chest, and many other physical conditions perfectly independent of disease of any of the thoracic organs, modify the extent of propagation so variously, that there can be no practical utility in laying down rules subject to perpetual exceptions. But the lines of propagation of the two sounds severally agree in most healthy persons, whatever be their absolute intensity at their seat of production; changes in these lines point positively to some modifying cause, and hence their establishment is clinically valuable. Now, the first sound passes slantingly upwards to the left acromial angle, growing weaker and weaker on the way; it loses much more on the route to, and at, the right acromial angle: its propagation backwards is clearest and fullest to the left,—so that while audible at the left back, it may be inaudible at the right. The second sound, with the base-region as its centre, radiates to the right and left acromial angles, with greater clearness to the left than the right; posteriorly it reaches the surface at the right side less clearly than at the left.

401. The difficulty of unravelling the mechanism of the heart's

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* Some authors speak of the sounds being audible in healthy male adults, of moderate stoutness, even *at the right side posteriorly;* others write that the space over which they are heard *seldom exceeds two square inches in the cardiac region!* The truth is that both of these states exist in different individuals in perfect health: the error lies in regarding either of them as constant.

sounds is emphatically proved by the fact that, from the time of Laennec to the present day, at least thirty-one theories have been proposed in its explanation. It would be a tiresome, and indeed useless, task to review these theories *seriatim:* the better plan seems to be to state with as much brevity as possible, the rationale of the sounds, which is supported by the greatest amount of clinical and experimental evidence.

402. *First Sound.*—The phenomena, which are at once conceivably soniferous, and coincident with the heart's first sound, may be enumerated as follows:—*At and immediately after the commencement* of the sound, the impulsion of the blood in the ventricles against the auricular valves; the sudden tension of these, and the sharp collision of a portion of their surfaces; the attrition of the blood-elements *inter se* within the ventricles, and their impulsion against the ventricular walls; the projection of blood from the ventricles against the orifices of the pulmonary artery and aorta, and perhaps against the columns of blood contained within them, with the simultaneous flattening of the sygmoid valves against the arterial walls, and the diastolic extension of those walls; the shock of the heart's apex against the side, or against lung-substance, if this be interposed; and, finally, the attrition of the pericardial surfaces near the apex;—*towards the close* of the sound, the collision of the surfaces of the ventricles, after the expulsion of their contained blood; and *throughout the entire duration* of the sound, the sustained muscular contraction of the walls of the ventricles, together with, at an instant of systolic time varying with the energy of the heart's action, the perfect, completed tension of the muscular fibres.

Now, of all these possible elements of sonorousness, which actually contribute to the generation of the first sound? It seems to me that, if we except pericardial attrition (inasmuch as the normal first sound has no shade of friction-quality in it*), and, probably, collision of the blood-elements among themselves within the ventricles (inasmuch as experiments seem to show that such collision is, at the least, rarely soniferous), all these phenomena are more or less constant, and more or less powerful, causes of the sound. That the sound derives its dull, booming prolongation from muscular contraction, seems unquestionable, not only because it retains these characters when the heart contracts after separation from the body, and the action of the auricular valves is prevented, but because it may in these characters be pretty closely imitated by the contraction of voluntary muscles.* Nor can there be any doubt that the tension and surface-collision of the auricular valves, and sharp shock of the blood against their ventricular surfaces, give the comparative sharpness to the first sound,—a character which may be detected by attention at its outset, and which, in certain states of altered contractility of the muscular fibres, almost covers or rivals in strength the sound generated by these: the elements of sonorousness of a certain quality, exist in these conditions; and, when they are experimentally interfered with, a corresponding change follows in the character of the first sound. The projection of the ventricular blood against the orifices of the large vessels, the flattened valves, and the bases of the columns of blood they contain, combined with the sudden

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* This is, in my mind, a much more conclusive reason for its rejection than the fact that the first sound continued, not *obviously* changed, after the

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pericardium had been removed in living animals. Such continuance would merely prove that pericardial attrition took no *prominent* part in generating the sound. But in exceptional cases (where there is no reason to believe the pericardium diseased), the first sound has a distinct, though slightly-marked, attrition quality. This was evident, for instance, in a woman (Roberts, U. C. H., Oct. 10, 1850,) with general moderate dilated hypertrophy in whom the knock of the heart against the side was *occasionally* distinctly soniferous, and accompanied with a sensation of faint rubbing palpable to the hand: all three signs disappeared under rest and appropriato treatment.

* In a remarkable case, which I saw with Mr. Hardwicke, of hypertrophy of the recti abdominis, attended with powerful contractions, partly reflex, partly consensual, of their tissue, the variation in intensity of tho muscular sound, according as the contractions were slow and gradual, or quick and abrupt, was very striking: in the latter case, the sound had a very distinct resemblance to the first sound of the heart.

extension of the arterial coats beyond, have strong clinical and experimental claims to a share in the first sound. A sound is audible in the arteries, synchronously with the heart's systole, under circumstances in which the idea of mere conduction from the heart is quite inadmissible: such sound may be heard in the femoral and even popliteal arteries sometimes, where no disease of these vessels or of the aorta exists. Again, in certain cases of mitral regurgitant disease, where true systolic sound at the left apex is completely deficient (a murmur only existing there), the first sound may be discovered with much of the quality of health at the base. There is a want of clear evidence that the collision of the surfaces of the ventricles normally contributes to the production of the sound at its close, especially as the blood of the ventricles is not completely expelled by their systole: but probably in some morbid states it may do so,—after profuse hæmorrhage, for example, where the clicking character of the sound seems thus fairly explicable. Lastly, the heart's shock against the side, especially when the posture of the individual, the period of the respiratory act, or other conditions, allow the surface of the organ to play fully against the parietes, indubitably increases the first sound, and gives it in particular cases a knocking character.*

The essential causes of the first sound, then, seem to be muscular action (that of the walls of the ventricles), valvular tension (that of the auricular valves), and forcible shock of fluids against resisting membranes (that of the blood against the orifices of the large vessels); while certain subsidiary causes act occasionally, especially impulsion of solids against solids (that of the heart's apex against the chest walls).

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* Where a thick layer of emphysematous lung intervenes, and sometimes even without this, I think there is reason to believe the first sound may be given an *intermittently murmur-like character* at the apex, independently of any disease in the mitral valve, simply by the apex-point of the heart impinging against the lung, and moving some of its contained air. I have observed the phenomenon, during suspension of the respiration, where this seemed its sole plausible explanation.

403. *Second Sound.*—The possibly soniferous phenomena, synchronous with the second sound, are: the diastole of the ventricles, and rush of blood into their cavities; the sudden recedence of the heart's apex from the chest walls; the abrupt fall of the auriculo-ventricular valves to the sides of the ventricles; the sudden tension of the sygmoid valves, and impulsive fall of the columns of blood against them during the arterial systole; the arterial systole itself.

Now, of all these causes, the most effectual seems to be, as originally taught from clinical observation by Sir R. Carswell,* the tension of the sygmoid valves: the absolute disappearance of the natural second sound at the aortic orifice, and its persistence at the pulmonary orifice, in cases of insufficiency of the aortic valves, is a sufficient proof of the fact. The quality of the sound, and the site of its maximum force, as already described, depose, too, in favour of its membranous origin, and of its localisation at the orifices of the great vessels.† The fall of the columns of blood on the surface of the valves, (though not so sonorous as if the valves, instead of being opened out by the receding blood, as they are, were *first* expanded, and *then* received the shock of the fluid from above, as is affirmed to be actually the case by M. Hamernjk, who but revives an opinion of Lower,) must intensify the sound of valvular tension. Whether the arterial systole itself plays any part in its production, is yet open to inquiry.

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* See Archives gén. de Méd., t. xxvi., 1831.

† The experiments made on large animals leave the mechanism of the second, and, *à fortiori*, that of the first, sound far from satisfactorily established: the matter really rests on clinical evidence. Thus, in Hope's records of experiments in which both pulmonary and aortic valves were hooked up, the simple statement is made, that the "natural second sound entirely ceased, and was replaced by a prolonged hissing." (Dis. of Heart, Ed. 3, p. 35.) We are left in the dark as to whether the second sound was thus ascertained to have become inaudible at the base only of the heart, or over its entire surface. From several passages in the context, the former seems the more likely; and hence the records at least of these experiments leave it still an open question, whether or not the natural second sound is normally, in any small degree, ventricular in site, or capable of becoming so, when aortic regurgitation exists.

Powerful though the diastole of the ventricles be—sufficiently so even in the new-born infant to open out violently the hand closed around them *—it seems certain, as matter of experiment, that the phenomenon is soundless. Skoda argues, that as exceptional cases occur in which the second sound is weak at the base, and loud and clear at the apex, while there is no diastolic impulse against the chest wall, the sound must originate in part in the ventricular region. He suggests that it may be produced either by, first, the stroke of the blood under special circumstances against the ventricular walls during the diastole of the heart: or, secondly, by separation of the heart's apex from the surface, against which it had been pressed during the systole; or, thirdly, by separation from the chest-wall of the portion of pericardium which had been driven against this during the systole. I am disposed to believe that there may be more truth in the two latter notions than there seems on surface consideration: if the chest be percussed with the point of a finger, while the ear is applied to a solid stethoscope in a neighbouring spot, two sounds are heard for each blow; the first strong, corresponding, of course, to the direct impulse; the second, very weak, to the removal of the finger from the point percussed. Now the recedence of the heart's point from the side is here imitated. As regards the first hypothesis, in the normal state, the blood enters the ventricles from the auricles, with a current so calm as as to render it singularly unlikely that audible sound can be thereby produced in their former cavities. But in cases of highly marked aortic regurgitation, blood falls with notable force into the left ventricle, and may conceivably generate sound. I have unquestionably heard, at the left apex, a distinct sound in more than one such case, while at the aortic base the ordinary regurgitant murmur alone

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* As ascertained by Cruveilhier in his case of Ectopia cordis.

existed: such cases would probably be more frequently met with, were it not for the loudness, and transmission to the apex, of the murmur evolved at the base.* I am persuaded this intensified second sound cannot be the transmitted sound of the pulmonary valves, because I have found it stronger at the left than the right apex.†

Although diastolic jogging impulse may occur without actual disease of the heart, yet there is no proof of such impulse being *per se* soniferous; the more so as, even in cases of strong morbid diastolic impulse, that impulse does not distinctly produce sound. M. Magendie's idea that the normal second sound is produced by the shock of the right ventricle against the sternum is, in my mind, illusory. Again, the fall of the auricular valves against the ventricular surface is not demonstrably sonorous.

Hence the sudden tension of the sygmoid valves, and the fall of blood upon them, seem in the *normal* state the sole demonstrated causes of the second sound.

MODIFICATIONS OF THE HEART'S SOUNDS OCCURRING INDEPENDENTLY OF CARDIAC DISEASE.

404. The description now given of the sounds of the heart applies especially to the male adult. The first sound is clearer and more ringing in females and in children, hence apparently more intense, and, *cæteris paribus*, more extensively audible.

405. Variation of posture has little, if any, effect on the second sound: but the first is commonly stronger in the erect than in the reclining, and in the prone than in the supine, postures: the reasons are obvious.

406. The extent and direction of transmission of the healthy heart's sounds are modified by various changes in the conducting

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* Flood, U. C. H., Males, vol. vii. p. 265.

† In a certain proportion of cases of mitral regurgitation with dilated hypertrophy, an intensified second sound may be found in the left apex;—its explanation will be sought hereafter.

power of the contents of the thorax [288]; unless the absence of such changes has been established, we are not justified in inferring that a plus or minus extent of propagation depends on the state of the heart itself. Enlargement upwards and inwards of the liver or spleen renders the sounds unduly audible in the hypochondria.

407. When the heart is displaced by diseases extraneous to itself, it might be supposed that the points of maximum force of its sounds would likewise be displaced: this is true, however, to a much greater extent of the first sound than of the second, and for the evident reason that the apex of the organ is more moveable than its base. In left pleuritic effusion, for example, when the maximum point of the first sound is carried to near the right nipple (say seven or eight inches out of its place), the maximum of the second scarcely swerves farther than to the right edge of the sternum, or one and a half inches from its post. Now it follows from this, that the line connecting the maxima points of the two sounds deviates, more or less, from the almost vertical bearing it presents in health.

408. The intensity of the heart's sounds, and even the pitch of the first, is heightened by nervous excitement of all kinds: in the hysterical and epileptic paroxysm this is sometimes strikingly remarkable; emotion, whether of fear, anger, &c., has a similar effect, the sounds being audible even to the individual himself, and to by-standers at one or two feet distance from the chest. Diseases of debility weaken both sounds; but this effect is greatly more perceptible in the first than the second: the causes of the first sound give the clue to this difference.

409. Certain conditions change the combined force and quality of the first sound by influencing some one of its elements without affecting the others. In continued fever, the general weakness, impairing the muscular power, throws the valvular element into undue prominence, and gives the first

sound a clicking character, akin to that of the healthy second. Again, nervous excitement intensifies and gives a ringing quality to the impulsive portion of the sound, so much as to throw the others into the shade. The second sound is comparatively little affected in either case.

410. These changes occur at the spots of production of the sounds. Can the heart's sounds be increased in force, or changed either in pitch or in quality, subsequent to their generation, and through the influence of conditions beyond the heart?

I have known both sounds materially louder in the site of a cavity under the left, more rarely the right, clavicle, than at the mid-sternal base. More than this, in a case of well-marked chronic consolidation of the apex of the right lung, *the second sound of the heart* (this organ, the arch of the aorta, and the pulmonary artery being, as far as ascertainable from signs and symptoms, perfectly healthy) *was decidedly louder under the right clavicle than at the mid-sternal base.* No such reinforcement existed at the sternal notch, nor under the left clavicle; the mediastinum and left apex were, judging from their percussion-note, free from solid matter. Here, then, was a case where the second sound was louder at about three inches' distance from its point of production than at that point itself: the pitch of the sound in the two places was identically the same.* Was the reinforcement produced on the principle of echo, consonance, or unison-resonance?

Of change of pitch I know nothing by experience. But the condition of the hollow organs of the abdomen, especially the stomach, sometimes curiously modifies the quality of the heart-sounds. When that viscus is distended with gas to a certain amount, the sounds echo within it with a metallic ring, and so loudly, sometimes, as to be perceptible to, and seriously alarm, the patient.* Large cavities in the adjoining lung (especially if their walls be hard, and the pleural sac distended with gas,) sometimes echo the heart's sounds in a similar way.

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* In Allen, U. C. H., Males, vol. ix. p. 196, the same phenomenon was ascertained on the left side.

411. The number of sounds corresponding to each revolution of the heart may be increased, but not diminished, independently of structural disease in itself, in the manners to be presently described.

MORBID STATES DEPENDENT ON HEART-DISEASE.

412. These are referrible to the heads of — I. Modified sounds; and, II. Adventitious sounds or murmurs.

413. I. *Modified sounds.*—The intensity or loudness, and the extent of transmission of the sounds, when modified by heart-disease, are in the direct ratio of each other. *Increase* in both respects is observed to its maximum in dilated hypertrophy, the valves remaining sound; muscular substance exists in sufficient excess to intensify the muscular share of the first sound, while the thinness of the walls, as compared with the size of the whole organ and capacity of its cavities, acts as a special source of loudness. The forcible propulsion of the blood into the aorta leads to proportionally forcible reaction of the arterial coats, and unusually sharp recoil of the column of the blood on the sygmoid valves,—hence a powerful second sound. In simple dilatation, the first sound, louder than in health, is sharper and clearer also: the heart is brought more extensively than natural into contact with the chest walls, and the thinness of the muscular walls allows of short abrupt contraction. Induration of the walls of the heart weakens the muscular portion of the first sound, but intensifies the valvular; and, if the other conditions for this exist, gives peculiar loudness to the impulsive noise produced by the apex.

Functional excitement of the organ also intensifies the sounds, whether this be dependent on inflammation of the organ itself or of its membranes, or on general febrile disturbance previous to the occurrence of debility. But nervous excitement is the most powerful intensifier of the heart's sounds ; it will even temporarily produce a loud first sound from a pure solidly hypertrophous left ventricle, and taken alone has much more frequently been known to render the sounds audible at a distance from the surface than any organic affection of the heart. I doubt if the latter, unaided by nervous excitement, be competent to produce the phenomenon.

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* Warren, U. C. H., Males, vol. viii. p. 35: aneurism of arch of aorta.

The quantity of blood circulating through the heart also modifies the intensity of the first sound. If the quantity be considerable, the struggle of the ventricle to force it on is considerable and prolonged ; but the sound does not gain, indeed rather loses, in loudness. When the quantity is small, on the contrary, there is no such struggle ; the ventricles contract abruptly and sonorously, and probably, under these circumstances, collision of their opposite surfaces intensifies as well as modifies the quality of the first sound.

414. The sounds are *weakened* where the muscular structure of the heart is encroached upon by morbid infiltrations, or by its own disease. Softening, obesity, fatty degeneration, cancerous infiltration, atrophy with fibrous infiltration, all impair its force, both in its muscular and valvular elements, for obvious reasons. In dilatation with attenuation, the first sound, though clear, is feeble,—probably from conjoined change in the sarcous structure.

In simple, and still more in concentric, hypertrophy of the left ventricle, the first sound is weakened at the apex in, it may almost be said, the direct ratio of the increase in mass of the muscular substance, and decrease of the cavity. In extreme cases, the first sound may be completely deficient over the ventricle, and perceptible only at the base, or towards the ensiform cartilage. The compact packing of the muscular structure interferes probably with the freedom of vibration necessary for the generation of sound, and the diminished size of the cavity obviously impedes the play of its parietes. Even valvular action is diminished in scope, and while the mitral valve is of less proportional extent, it is often thicker than natural. The entire mass of the ventricle, too, does not contract simultaneously.

Accumulations in the pericardium of fluid, of air, or of air and fluid combined, weaken the sounds by removing their seats of production from the surface. I do not know clinically the effect of solid formations in the pericardium; they may be conceived to weaken the sounds in one way, and intensify them in another,—severally in production and in conduction.

Want of muscular and nervous tone, generally, weakens the first sound, as well as modifies its quality.

415. The *duration* of the first sound falls below the natural standard in dilatation without hypertrophy, or with attenuation, and under all circumstances which weaken the muscular, and throw into undue prominence the valvular, portion of the sound. The sound is lengthened more or less in hypertrophy, with moderate dilatation; and if, in addition, the mass of circulating blood be large, or if the aortic orifice be at all obstructed, the sound may be sufficiently prolonged to fill nearly two-thirds the period of each revolution of the heart.

The second sound is shortened by a thin papery state of the sygmoid valves and by thinness of the blood; its pitch rises under the same influences. The sound is lengthened, on the contrary, by thickening of the valves, and a loose inelastic condition of their texture. Probably, too, thickening of the non-striated muscular portion of the arterial walls will lengthen their own systole, and hence, probably, the diastolic heart-sound. Another curious cause of prolongation of this sound will be by-and-by described.

416. The points of maximum intensity of the heart's sounds are more liable to displacement by extrinsic than by intrinsic causes; the latter, however, in some forms influence them. Enlargements of the heart generally, whatever be their nature,

lower the maximum point of the first sound. Simple, and *à fortiori* concentric, hypertrophy influence its position much less, in proportion to their mass, than the eccentric. Pericardial effusion, raising the apex, *raises* the point of maximum intensity of the first sound; and in some rare instances I have known this carried backwards, the sound being more distinct in the left vertebral groove than in the ordinary apex-region. Hypertrophy of the right ventricle lowers its own first sound; of the left, carries its own immediately to the left, as well as depresses it. In all cases of depression of the first sound, the second is similarly affected, but to a less degree. Marked eccentric hypertrophy of the auricles would also probably lower the first more than the second sound; but I do not know this from experience.

417. The distance of the heart's sounds from the ear of the auscultator is very manifestly *increased* in cases of fluid and gaseous accumulation in the pericardium: but there is a source of fallacy here; the sounds are commonly listened to in the natural situation of the apex, and not in the neighbourhood of the nipple or fourth rib, whither it has been carried by the disease. Agglutination of the pericardial surfaces, on the contrary, brings the sounds *nearer* than natural to the ear; if not precisely at their maximum points, beyond these. Enlargement of the heart, sufficient to push away the left lung, will have a similar effect. The first is more affected by these changes than the second sound.

418. The quality, and with this the pitch, of the heart's sounds is subject to serious modifications. The first becomes dull, muffled, toneless, and in some cases almost null, at the apex, where dense hypertrophy is conjoined with a thickened inelastic condition of the auricular valves. On the other hand, where the ventricular walls are thin and the valves natural, the first sound becomes more or less clear, flapping, or clicking, with raised pitch; if those walls be in a state of eccentric hypertrophy, and the valves somewhat thickened, the sound

assumes a clanging character. At least, these statements are in accordance with the majority of results; but exceptions, explicable, perhaps, sometimes by the state of the heart's texture,—in others inexplicable,—pretty frequently occur. It would be difficult to describe or explain, for instance, the varieties of quality found in the first sound of soft, flabby, fatty hearts, with healthy, or nearly healthy, valves. Clearness and elevation of pitch depend, as a rule, either on thinness of the muscular walls, or on predominance of the valvular element of the sound.

The first sound sometimes possesses a peculiar full-toned quality, without the least sharpness, while it is strongly accentuated at the commencement, and commonly prolonged: the nearest articulate symbol of the two sounds, under these circumstances, appears to be *b'oom-tup*, pronounced with strong emphasis on the *b*. I have observed this peculiarity in cases of eccentric hypertrophy of the left ventricle,—but without ascertaining the special condition on which it depends.

The quality of the first sound at the apex is sometimes sharply knocking; but with care this knocking quality is separated by the ear from the true heart-sound, and obviously depends on the impulse of the apex against the side,—but not necessarily against the inferior border of the fifth rib, as imagined by Dr. Hope. Knocking impulsive sound cannot be called an essential or even habitual attendant on any particular disease of the heart; nervous palpitation, especially if the edge of the lung be by some disease of its own carried unduly towards the left, will readily produce it in a healthy organ. Thin-walled resonant chests supply it with greater ease than others; and morbid induration of the heart's apex, or calcification of the pericardium, will aid in intensifying it. The heaving and steadily pushing character of the impulse in simple hypertrophy prevents its occurrence in that disease: eccentric hypertrophy is the form of enlargement it most frequently accompanies.

419. The *first* sound is sometimes slightly rough, and approaching in quality to a murmur at the apex; it is, in fact, *murmur-like*, without being actually converted into a murmur.* This may be (*a*) a persistent condition, observable week after week, while the patient remains under treatment; or (*b*) a temporary state, constantly noticeable for a few days, and then disappearing; or (*c*) a mere transient phenomenon, occurring with some, absent from other, beats of the heart. In the first case (*a*), it has appeared to me referrible to an incipient or slight amount of some one of the organic conditions which, carried further, produce a perfect systolic murmur; or, probably, sometimes to a buzzing, murmur-like quality in the muscular sound itself, produced by slow contraction of the fibres, or some special alteration of their texture. The second case (*b*) is exemplified by some *excessively rare* instances of acute rheumatism, where the systolic sound at the left apex, roughened and murmur-like for a few days at the outset, then loses this quality, either permanently, or to resume it again at a later period in a more decided form.† Passing vascular roughness of the mitral, or even ventricular, endocardium, and imperfect closure of that valve from the influence of irritation, suggest themselves as possible causes of the phenomenon; but anatomical evidence is of course wanting on the point. In the third case (*c*), the peculiarity is caused either by coincidence of the respiratory sound with that of the heart,‡ or by rubbing of the apex against the pericardium,§ or by movement of air in the adjoining lung-substance produced by the cardiac

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* In the case already referred to [note, p. 213], when the contraction of the abdominal muscles occurred in a slow vermicular manner, the sound was distinctly of murmur-like quality.

† James Hayes, U. C. H., Oct. 1850.

‡ It is sometimes impossible to satisfy oneself on this point, unless by causing the patient to suspend his breathing.

§ The more superficial character and the influence of change of posture will generally distinguish this variety from a true murmur.

impulse, or (by far the most important cause, because the most likely to lead to error) by a tendency to reduplication of the first sound.

The *second* sound at the base is rendered dull, and comparatively clanging, by fibro-fatty thickening, without insufficiency, of the sygmoid valves. Diminished elasticity of the arterial walls has a similar effect.

Like the first, the second sound may be murmur-like, temporarily or permanently. Very trifling insufficiency will probably thus modify its quality; whether marked reticulation of the valves will suffice for the purpose, will hereafter be discussed. The most common cause of murmur-like quality in this sound is a tendency to reduplication.

420. The natural *accentuation* of the sounds, as shown in a previous page, is liable to numerous perversions; but as the accent falls on whichever sound is intensified, and mainly at the spot of intensification, repetition may be saved by referring the reader to the paragraphs on augmented intensity. When the heart is weak and flabby from organic change, or from want of tone, there may be a total deficiency of accent on either sound at the apex;* the sounds resemble those of a vibrating pendulum.

421. Like the sounds, the *silences* vary in disease in relative duration. The first, or post-systolic, silence is lengthened by deficient elasticity of the arterial walls, whereby the recoil of the blood on the valves is sluggishly effected; so, too, whenever the first sound is disproportionately shortened, the first silence is lengthened. The first silence is normally so short, that it is difficult to appreciate its decrease.

The second, or post-diastolic, silence is lengthened in cases of advanced constriction of the mitral orifice; the process of filling the ventricle is laborious and slow, and hence the systole lags, as it were, behind its time. When the circulation is greatly slackened, the second silence is generally disproportionately prolonged.

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* The second sound is, in certain rare cases of mitral regurgitation, so intensified that the accent falls on it, even when auscultated at the apex.

422. The relationship of the sounds of the heart to the pulse varies in disease. In the normal state, the first sound is apparently synchronous with the diastole of the arch of the aorta, the pulmonary, carotid, and subclavian arteries; thenceforth, the further the vessel from the heart, the more distinct is the interval between the systolic sound of that organ and the arterial diastole. It is difficult to determine the possible length of interval consistent with health: but it may be affirmed, that if the diastole of the most distant vessels, as the posterior tibial and dorsal artery of the foot, is so much retarded as to become synchronous with the second sound, the state is morbid. This retardation, which was first noticed by Dr. Henderson as an attendant on insufficiency of the aortic valves, may with care be detected in many, but unquestionably not in all, cases of that disease. Possibly where no morbid retardation can be discovered, the failure may depend, not on its absence, but on its being carried to such extremes, that the arterial pulse produced by one cardiac systole is nearly synchronous with the next. The only fact, however, I know of, supporting this idea, is, that it is in *extreme* cases of aortic regurgitation that the pulse seems occasionally to stand in normal relationship of time to the heart's systole. The same sign exists in attenuated dilatation of the left ventricle also. Again, in health, the frequency of the pulse and the length of the systolic sound vary inversely as each other: a frequent pulse is the index of a short first sound, and *vice versâ*. The same relationship holds good in some morbid states; for example, in anæmia and in the re-action after hæmorrhage. It is, on the other hand, occasionally perverted; the pulse may be infrequent, and the systolic sound short: in fatty degeneration, and in simple flabby softening of the heart, this perversion may sometimes be noticed.

423. The sounds of the heart are sometimes suspended for the

precise length of time occupied by an ordinary revolution of the organ: they are said then to *intermit.* Not uncommonly such intermission recurs with considerable regularity; that is, after a fixed number of regular beats. Sometimes the systolic sound seems to *anticipate*, sometimes, on the contrary, to *hesitate* at, the proper moment of its occurrence,—changes of rhythm closely connected with shortening or prolongation of the second silence. Sometimes a series of feeble and rapidly succeeding sounds follows others comparatively loud, slow, and deliberate; and there may be a certain uniformity in the number of each kind, and in the periods of their recurrence. Or the *irregularity* of the sounds may be complete, both in intensity and in rhythm, no two revolutions corresponding to each other in either character: there ceases to be any semblance of order in disorder. This excessive perversion exists in highly marked mitral contraction and regurgitation, in extreme softening, fatty infiltration, acute destruction of a portion of a valve, or of chordæ tendineæ, rupture of these structures, formation of fibrinous coagula within the heart, and in a small proportion of cases of pericarditic effusion.

424. The natural correspondence in the number and time of cardiac systoles and arterial pulsations is habitually maintained, even when the rhythm of the heart's contractions is thus variously altered. If the left ventricle intermits, or anticipates, or hesitates, or becomes wholly irregular in its contractions, a precisely similar change occurs in the arterial pulses: the impulses of the connected tubes are the counterparts of those of the central organ. But, on the other hand, there may be a failure of this correspondence, not only when the heart's contractions are thus abnormal in rhythm, but even when they are in this respect normal. Thus two revolutions of the heart may correspond to a single radial pulse, the cardiac action and the pulse being perfectly regular in force and rhythm; or the pulse may be perfectly regular, and the heart's successive systoles somewhat unequal in force and duration, as in a case formerly

recorded,* where eighty-eight systolic contractions produced forty-four radial pulsations. Here the rhythm of each pair of beats of the heart might be represented thus:—Systole = 5, diastole = 3; systole = 9, diastole = 4. It was the first of the two systoles that failed to affect the pulse at the wrist; and as there was no evidence of aortic or mitral disease, but merely of flabby enlargement, that systole may have been simply too weak to influence the distant vessels: the state was of temporary duration. I have observed a similar condition, persistent, but of less regular type, in cases of extreme contraction of the mitral orifice: under these circumstances, doubtless the systole occasionally takes place before the ventricle is supplied with blood to propel.

Again, in certain cases of utter irregularity of the sounds, there may be no traceable accordance between them and the force or rhythm of the pulse. This is, perhaps, best observable where the irregularity comes on suddenly from rupture of a valve, or accumulation of coagula in the cavities: but is occasionally met with in all the diseased states productive of irregularity.

425. The *number of sounds* attending each beat of the heart may vary, the arterial pulse holding its natural relationship to the systole. A *single* sound only may be heard, and this may be the first or the second; whichever sound be deficient in any particular spot, it may, or may not, be audible at some other part of the cardiac region. The first sound may be quasi-deficient at the left apex, when the conditions, already described as weakening it, are carried to extremes; but it will then be found at the right apex, and at the base. So, again, the second sound may be quasi-deficient at the base from excessive feebleness, or from being covered by a prolonged systolic sound, or systolic murmur: but in the first case, excitement of the heart, increasing the energy of its contractions, will invigorate the sound, and in the

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* Clin. Lect., loc. cit. p. 443.

second case, the sound will be heard at the right apex. Absolute deficiency of either sound, or of a murmur taking its place, has never fallen under my observation; in other words, neither systole nor diastole has ever been, in my experience, absolutely noiseless over the entire cardiac region.

426. The systolic or diastolic sound, or both of them, instead of being single may be double; the affected sound is then said to be cleft, doubled or reduplicated. The phenomenon is far from uncommon in some of its forms.

Such slight accentuation as exists falls usually on the first of the three or four sounds; their pitch is sometimes distinctly different. Reduplication existing at one spot may be audible elsewhere, or limited to that spot. The forms of reduplication, which have fallen under my notice, are the following:—

First sound.—Double at the left apex (*ŭbbŭp-dŭp*), at the same time single or double at the right apex, and single,* or, more commonly, double, at the base.

First sound.—Double at the right apex (*ŭppŭp-tŭp*), single at the left apex and at the base.

First sound.—Perfectly doubled at the base, imperfectly so at the tricuspid apex, single at the mitral apex.†

First sound.—Double at the base, or at the apex, while the second sound is conversely double at the apex or the base.

Second sound.—Double at the base (the most common of the series), very generally single at the apex; the reduplication audible or inaudible at the pulmonary and aortic cartilages, or at either singly.

Second sound.—Constantly double at the tricuspid apex, and single at the base and left apex;‡ or, while constantly double at the tricuspid apex, occasionally so at the base.§

First and second sounds.—Both reduplicate (*ŭppŭp-tŭrrŭp*). This is very rare, is more frequent at the left apex than elsewhere, and has never appeared to me audible at all parts of the heart's area.

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[*] Bowry, U. C. H., Males, vol. vii. p. 259.

[†] Branchley, U. C. H., Females, vol. ix. p. 94.

[‡] J. Hayes, U. C. H., Males. Oct. 17, 1850.

[§] W. Hodson, U. C. H., Males, vol. ix. pp. 65, 66.

Reduplication may change from one sound to another.* Doubling of the second sound at the base may be arrested by deep inspiration; or the second sound at the pulmonary cartilage, single in expiration, may be most distinctly split into two at the end of inspiration.† Sometimes, again, a tendency to reduplication exists without the sound being actually double; this state of things prolongs, and may give a murmur-like quality to, the affected sound.

427. The essential cause of these various reduplications seems to be a want of synchronism between the actions of the two sides of the heart. If the facility with which the two ventricles fill with blood be unequal, they will probably differ somewhat in their time of contraction; certain conditions of the auriculo-ventricular valves may bring their closure on either side, as it were, behind time; the production of a double systolic sound at the apex becomes thus readily intelligible: but the fact that such reduplication may be audible at one apex only, and perfectly inaudible at the base, is far from being easily explicable. It appears, too, from a case observed by M. Charcelay,‡ that the contraction of the auricles, when highly hypertrophous, may become sonorous, and so double the first sound; but it is scarcely necessary to add, that such mechanism is of singular rarity; nor does it appear probable that the characters of the reduplicate sound could, under the circumstances, resemble those ordinarily met with.§ A simulated reduplication of the first sound may sometimes be produced by the knock of the heart against the side; but the least-practised ear

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* Branchley, U. C. H., Females, vol. ix. p. 146. "At base first sound double, when first listened to; within two minutes the second reduplicated: no doubling of either sound at left apex during examination."

† S. Roberts, U. C. H., Females, vol. v. p. 220.

‡ Archives générales de Médicine, 1838, p. 393.

§ I have known an hypertrophous left auricle produce a distinct, even loud *knocking* sound at the third left cartilage.—Fred. Smith, U. C. H., Males, vol. v. pp. 297-8, Nov. 1850.

will readily distinguish this from true *intra-cardiac* reduplication.

Non-synchronous tightening of the sygmoid valves, again, easily explains the reduplication of the second sound at the base; and may itself be referred to unequal elasticity in the coats of the aorta and pulmonary artery, stiffness of either set of valves, a material obstruction in the way of their closure, or any state of either ventricle rendering it slightly tardy in the propulsion of its blood into the artery beyond. But the same difficulty re-appears in accounting for the limitation of the phenomenon to certain points of the cardiac region.

428. In regard of diagnosis, it must be confessed, these reduplications are almost insignificant in the present state of knowledge. And for the following reasons :—reduplication is never, as far as I have observed, permanent and invariable; it occurs most commonly in hearts either healthy or temporarily disordered in function only : less commonly in cases of slight organic affection; and with least frequency when serious valvular disease exists;* it is not connected, as a rule, with any particular form of disease; it comes and goes in the course of a few beats of the heart; sometimes disappears on change of posture, and is affected even by the act of respiration. The real interest of reduplications arises out of their bearing on the theory of the heart's sounds,—a fact of which the following illustrations are sufficient evidence.

The second sound may be continuously doubled at the base, and perfectly pure and single at the apex. How is this explicable on the simple sygmoid theory of the second sound ? A double sound does not become single by conduction over so short a space.

The first sound may be single at the left apex and at the base, while it is distinctly reduplicate at the right apex. Here the ventricular and arterial portions of the first sound seem to be separated on the right side of the heart.*

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* It is fair to observe, however, that the existence of murmurs in such cases may be the real cause of reduplication not occurring, or not being heard.

The second sound may be double at the base, and single at the aortic, double at the pulmonary, cartilage or *vice versâ:* This cannot arise from want of synchronism of the two sets of valves, but of the three divisions of one set.

The second sound may be single at the base and double at the left apex; now, according to the pure sygmoid theory, the arterial valves are the sole source of the second sound: how come the two sets to divide their compound sound at the apex? Splitting into two, as a result of conduction from the base to the apex by ventricles of different conducting powers, cannot be admitted; for the reduplication may be present at the left apex only, absent at the right. This is the strongest fact, I know of, in favour of the second sound being in some cases partly of ventricular origin.

429. II. *Adventitious Sounds or Murmurs.*—Sounds of adventitious origin and properties produced either within or on the surface of the heart, are termed Murmurs; according to their seat of production, they are divided into Endocardial and Pericardial.

ENDOCARDIAL MURMURS.

430. The *special character* of all endocardial murmurs is, more or less, blowing. Their quality varies extremely, and they may be called simply blowing, grating, filing, rasping, sawing, whistling, or cooing, according to their greater or less similarity to these sounds. The simple blowing murmur, though itself presenting different degrees of harshness, is always soft in comparison with the filing, grating, rasping, and sawing varieties. In some cases they are tones, capable of musical notation. Endocardial murmurs rarely become audible to the individual himself [433]; they may in some instances be heard at

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* In one case (James Hayes, U. C. H., Oct. 17, 1850) where this form of reduplication was well marked, pericardial adhesions had recently formed.

short distances from the chest.* They are essentially *intermittent;* and, no matter what its duration be in relation to the heart's contractions, a single murmur is never sustained continuously, either in a uniform or remittent manner, through a series of beats. In point of absolute duration, they vary from a scarcely appreciable moment to two or three seconds: the latter amount of prolongation can only occur where the physical conditions are at once peculiarly favourable for sustainment of sound, and the action of the heart extremely slow. Their *pitch* varies by several notes, the lowest being, perhaps, represented by the whispered word *who*, the highest by *ss*; intermediate notes may be represented by whispering the word *awe* by inspiration, and the letter *r*, with various degrees of closeness of the isthmus of the throat: these are the suggestions of Dr. Hope. In point of apparent *distance from the surface*, they vary also: so deep, in some cases, as obviously to be weakened by distance; in others, they seem to originate directly under the integuments. The spots of *maximum force* of individual murmurs, the position of the heart being normal, are four,—a few lines above the left apex; just above the ensiform cartilage; at mid-sternum, on the level of the third interspace; and at the junction of the third left cartilage with the sternum. But these points of maximum force are liable to change, both from various mal-positions of the heart, and from alteration in the conducting qualities of the materials around it; the laws of transmission are then the same as in the case of the healthy sounds: a special cause, the direction of the current of blood, will be considered hereafter. An endocardial murmur once developed is habitually *persistent*, and attends every beat of the heart: however, weak systoles may fail to produce a murmur well marked with strong ones, tendency to syncope, general debility, and collapse, and the approach of death may prevent its production; and sometimes (for instance when the cause of the murmur is pressure on an arterial orifice by a tumor) certain postures may annul a murmur completely. Murmurs habitually attain a higher type of harshness the longer they exist, though a fall in pitch often takes place.

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* I have only once to my recollection met with this. In a case of cyanosis occurring in a child about six years old, a systolic rough blowing murmur could be heard at a distance of an inch or a little more from the surface.

430.* The *rhythm* of murmurs may be considered in respect of the heart's contractions and of the heart's sounds. In respect of the former, they are said to be systolic and diastolic; in respect of the latter, synchronous with the first or second sounds. But they are not necessarily synchronous with either systole or diastole. They may be pre-systolic, systolic, and post-systolic; pre-diastolic, diastolic, and post-diastolic: they may occupy a portion only of either time; or fill this and the succeeding silence, and encroach upon the succeeding sound,—as is common with systolic, rare with diastolic, murmurs. This arrangement is doubtless open to the charge of hyper-division; but as it positively has its foundation in correct clinical observation, it ought to be kept in view, although for ordinary purposes the simpler one is quite sufficient: there can be little doubt that many of the alleged failures of the rules for valvular diagnosis are traceable to inattention to these subdivisions of systolic and diastolic time. But murmurs are further divisible in respect of rhythm: whether systolic or diastolic, they may occur in the direction of the current of blood, or against it. Thus the systole of the left ventricle may produce a murmur at the aortic orifice with the current, or at the mitral orifice against it: hence a division of murmurs into direct and indirect,—which are also severally called, from their common causes, murmurs from constriction and from insufficiency of valves or regurgitation.

431. The effect of murmurs on the sounds varies. A murmur may simply render a synchronous heart-sound obscure, at its commencement, at its close, or throughout its entire duration; or it may completely mask this by its intensity, and even similarly affect the succeeding sound; or it may prevent the natural heart-sound from being formed. Thus at the left

apex a systolic murmur may completely drown a systolic sound, which is readily audible at the right apex and at the base. The systolic sound is not masked there, but really deficient, when both auricular orifices are not in a condition to produce natural sound, but murmur alone, and the arterial, or basic, portion of the first sound is feeble, or itself converted into a murmur.

When, as is very common, both murmur and sound are distinguishable coetaneously, the state may be called one of pseudo-reduplication. But true reduplication of a murmur is so excessively rare, that I remember to have met with but two examples of the fact: one, basic and diastolic, must have been in the aorta alone, for the pulmonary artery was unaffected: of the other, systolic at the left apex, I had no opportunity of examining the mechanism;—it might have been mitral and tricuspid together, or it might have depended on co-existing deep-seated venous hum,—in which case, of course, it was a false reduplication only. I once heard a post-diastolic basic murmur and sound in a rheumatic woman, in whom the doubling appeared to arise in consequence of the set of sygmoid valves, which gave the murmur, acting after the set which gave the sound.

432. Some murmurs are unaffected, others affected more or less seriously, by the posture of the patient. A systolic murmur limited to the left apex, and highly marked in the sitting or erect, may totally disappear in the supine posture, instantly re-appearing when the patient again sits up. Basic systolic anæmic murmur will often cease in the erect to return in the lying posture. Systolic murmur, strongest at the left apex, and powerfully marked in recumbency, may disappear in the erect posture, remain inaudible for several beats after the patient has again lain down, and very gradually recover its previous intensity.*

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* Constable, U. C. H., Females, vol. ix. p. 96. Here the patient was spanæmic, the aortic valves slightly diseased, the mitral not obviously unsound,—but whether regurgitation might or might not have occurred during life is matter of doubt.

Is this changeableness of a murmur proof of its inorganic nature? My experience would support the negative.

433. Patients, the subjects either of organic or functional cardiac disease, will occasionally describe noises, referred by them to the heart, and in which the physician at once recognises the characters of murmurs. Under these circumstances I have actually known endocardial murmurs exist of two kinds: systolic at the base, and systolic at the left apex. In the former of these cases the explanation seems easy; the murmur, transmitted along the aorta, reached the carotids. But in the latter case of mitral regurgitation, the explanation is not so readily forthcoming. In one instance of the kind, where there were neither carotid whiff nor jugular hum, but the mitral murmur was strongly reinforced by a flatulent stomach, I cannot refuse to believe that the patient actually heard that murmur, as she described its characters accurately, and, her heart never having been physically examined, until she was seen by myself, she could not, as it were, have learned her lesson from others.

434. When endocardial murmurs have existed during life, structural changes to explain them are found, or are not found, after death: hence their division into *organic* and *inorganic*.

435. (a) *Organic Endocardial murmurs* are essentially connected with such alterations of the orifices or of the valves, as, while they lead to constriction or imperfect closure of the orifices, cause unnatural friction between the blood and surfaces. The chief of these alterations are: simple *constriction*, or constriction with thickening, hardness, rigidity, calcification, warty or other excrescences from, or even simple inflammatory loss of polish and roughness of, the valves;—simple *insufficiency* of the valves to close a widened orifice, themselves not having grown *pari passu* with the widening, or such insufficiency depending on the various diseases of the valves just enumerated, or depending on shortening and thickening of the chordæ tendineæ, or on atrophy or contraction of the columnæ carneæ, or on puckering of valves, or adhesion of the divisions of a

valve *inter se*, or to the adjacent surface;—*excrescences* or other thickening or calcification of the valves, without either insufficiency or constriction;—*attachment of a tendinous cord* in a wrong situation, whereby it is thrown directly across the blood-current;—or *unnatural communication* between the different compartments of the heart, or between these and the arteries or some adventitious cavity. Besides, without distinct alteration of the orifices or valves, mere vascular roughness of the ventricular endocardium probably suffices to affect the purity of the sounds, when the current bears, especially towards the arterial orifices, on such a roughened spot. Coagula among the columnæ, or a polypoid body hanging from the neighbourhood of the valves, will have a similar effect.

Physically speaking, then, there appear pure *constrictions* of natural orifices, pure *widenings* of orifices, and pure *roughness* of surfaces, to explain the ordinary mechanism of organic cardiac murmurs. Now pure constriction and pure roughness are positively capable of producing murmur: this is matter of experimental demonstration; by pressing with the stethoscope on an artery, we convert the dull fillip-like natural sound of the vessel into a whiffing murmur. But how can pure widening of an orifice produce murmur? In the case of the tricuspid and mitral orifices, the *regurgitation* from such a physical cause may intelligibly produce murmur by the collision of direct and indirect blood-currents coming from and going back into the auricle: in the case of a *direct* current through a simply widened aortic orifice, the rippling of the stream, produced by the change of calibre, seems, from the experiments of Dr. Corrigan, sufficient, if that current be strong, to produce murmur.

436. Mere alteration in the direction of the current, of a kind to throw the blood obliquely against, instead of carrying it directly through an orifice, will theoretically generate murmur. Probably this divergence of the stream plays a part in many direct valvular murmurs: and as dilatation of the ventricles renders them more spherical and less convergent to their arterial

outlets, it has been urged by Dr. Blackiston and others that such state must produce murmur if hypertrophy co-exist,—that murmur actually is heard frequently under the circumstances,—and that, when wanting, its deficiency depends on the muscular energy being impaired by disease.* Hypertrophy with dilatation of a cavity, if its arterial outlet remain undilated, puts that outlet relatively in a state of coarctation: hence, too, may arise a murmur. I will return to these points with the subject of eccentric hypertrophy.

437. It was suggested by M. Martin-Solon that the pressure of the heart and great vessels by abundant pericardial effusion might cause murmur;—in a case of the kind, murmur, well marked in recumbency, disappeared when the patient stood up.†

438. The *properties of murmurs vary greatly with the conditions of the fundamental cause producing them*, and even with some conditions independent of this. Thus the force, loudness, or *intensity* of a murmur increases with the vicinity of its origin to the surface,—the density and hardness of the heart itself, and of the textures lying between the heart and surface,—the force and velocity of the current,—the amount of narrowing at an orifice,—and the volume of blood propelled through the obstruction. Excite a tranquil heart, and a murmur, previously, almost inaudible, becomes distinct; weaken the energy of cardiac contraction by digitalis or aconite, and the converse result follows.‡

The *quality* varies with the character of the surface over which the blood passes;—harsh and rough, if the surface be sharply uneven; soft, if smooth and merely constricted. But this influence is greatly modified, and may be actually reversed, by changes in the celerity of the circulation. The quality will also be materially affected by the condition of the intervening structures: if these be soft, the sound will be softened in quality; if hard, hardened. A musical quality is sometimes given when prominent spiculæ, of vibratile character, project into the current; and also when rigid vibratile edges bound a narrow chink-like opening.

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* Murmurs thus generated by *mis-direction of the blood-current* would really belong to the dynamic species.

† Journal Hebdomadaire, ix. 457.

‡ In a woman now under observation (Emma Powell, U. C. H., Dec. 1850), the systoles are so unequal in force that while some give a strong radial pulse, others produce none; the strong systoles are attended with systolic basic murmur, the weak ones with none.

The *duration* of murmurs will increase directly as the extent of surface in the condition to afford them, the amount of difficulty to struggle against, the quantity of blood, and the slowness of the circulation. If the structures intervening between the seat of murmur and the surface be imperfect conductors, the audible sound will be shortened,—its termination will be lost through imperfect conduction.

The *pitch* of a murmur is more under the influence of the size of the orifice through which the sonorous stream passes, than anything else: the smaller the orifice, the higher the pitch. But it is also raised by the tension of the walls of that orifice, and the thinness of the blood. The velocity of the current does not influence it, Dr. Blackiston urges, unless, as in blowing with a flute, the harmonic be elicited; but unless the edges of the diseased orifice be of such rigidity as to simulate the embouchure of the flute, it appears to me the two cases are not comparable.

The mere distance of the site of production of murmurs from the surface can in theory have no direct influence on their pitch. The same physical and dynamic conditions will generate sounds of the same pitch, whether they be close to, or as far as possible from, the chest-wall.

But may the pitch of a murmur change in course of conduction,—may the pitch differ, as audible at different parts of the parietes of the chest? To whatever distance a note continues audible, it remains (provided no intervening change of medium occur) the same note, as at the spot of its generation. But however difficult to understand, it does appear that where variation in media of conduction occurs, change of pitch is possible [270 (2)]; and as matter of experience I am persuaded that such change may be observed in cardiac murmurs. It is not uncommon to find the murmur of aortic regurgitation of lower pitch at the base, than at the tricuspid apex: it rises by conduction.*

439. Hence it appears that the properties of a murmur, as caught by the ear, are every one of them, singly, of complex mechanism,—and herein appears an easy clue to the absolute failure of all clinical attempts to establish the *precise amount and character* of the organic changes in a set of valves from the consideration of any one property, such as roughness, of the murmur they generate. Every necessary organic condition of a harsh murmur may be present, and yet the resulting murmur be soft, if the current be feeble,—nay murmur may be wholly deficient.†

440. (b.) *Inorganic Endocardial Murmurs.*—Endocardial murmurs, that cannot be traced to any organic cause after death, or that disappear so completely and permanently during life, as to preclude the idea of structural change in their production, are termed inorganic. They are divisible into two sub-classes—murmurs originating in some morbid condition of the blood (*hæmic*), or in defective action of the heart (*dynamic*).

441. *Hæmic Cardiac murmurs.*—An intra-cardiac hæmic murmur is of moderate or very slight intensity, commonly of

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* Flood, U. C. H., Males, vol. vii. p. 265. Case of highly marked aortic regurgitation: "the murmur tailed on to the second sound is higher in pitch at the ensiform cartilage than at the base . . . passing from the former point towards the left apex, it falls in pitch."

† I have, on the other hand, occasionally known a murmur, when audible in a calm state of the circulation, disappear during palpitation. I have only observed this in some cases of mitral regurgitation. Can it depend on irregular contraction of the wall of the ventricle allowing of such slight and feeble regurgitation that morbid sound cannot be generated?

medium or low pitch, short or moderately prolonged, of whiffing quality, very easily rendered temporarily harsh by excitement of the heart, and modified in intensity by certain changes of posture. This murmur is, as far as I have observed, invariably basic in seat and systolic in time, produced at the orifices of the aorta and of the pulmonary artery,—with a force at each proportional to the power of its communicating ventricle; scarcely conducted along the aorta at all; frequently audible, on the contrary, at the second left, or pulmonary cartilage; only in exceptional cases audible below the nipple; and never within my experience, perceptible as far as the left apex. The site and rhythm of hæmic murmurs, excluding all those of diastolic time and of seat at either apex, are of great value in their distinction. To their quality and pitch I attach but moderate importance; for organic murmurs may be soft, and inorganic ones are not very unfrequently rather harsh; while the latter may be shrill, whistling, and of high pitch, and the former are of course frequently of low pitch. Unfortunately there is no character in a systolic basic blood-murmur which positively proves its nature, and distinguishes it, *under all circumstances*, from one of organic source. The distinction is often rather to be made through the absence or presence of venous hum (I do not remember ever to have observed an intra-cardiac spanæmic murmur unattended with venous hum) and the course and duration of the phenomenon, than through its own immediate characters: *permanent* harshness and high pitch are never associated with true hæmic murmur. If arteries be the seat, extensively, of strong blowing murmur, on which the pressure of the stethoscope can have no influence, as, for example, the arch of the aorta, the innominate and subclavian arteries, a co-existing systolic murmur at the mid-sternal base is, in part at least, inorganic; but, on the other hand the cardiac murmur may be truly inorganic, and yet the arteries be perfectly free from abnormal sonorousness.

The morbid state of the blood most frequently associated with these murmurs is spanæmia,—whether it be that of chlorosis, of malaria, of starvation, of deficient insolation, of hæmorrhage and over-venesection, of carcinoma, of convalescence from acute diseases, &c. I have occasionally observed it in uræmia; so, again, it occasionally occurs, though to a very slight amount, in the hypinosis of continued fever and the exanthemata, and in the hyperinosis of pneumonia and acute rheumatism, under circumstances excluding more or less positively its dependence on endocarditis. It has been affirmed that plethora, rendering the quantity of blood too great for the cavities of the heart, produces murmur within the organ: confirmation of the statement is wanting; more especially if we agree with those chemists who maintain that the relative quantity of red corpuscles is increased in plethora.

The mechanism of these hæmic murmurs will be considered with that of the venous class.

442. *Dynamic Cardiac Murmurs.*—Under this head murmurs occurring in the heart, through some abnormal state of its action, range themselves. Violent excitement of the organ, whether it act merely by increasing the force of the current, or by disturbing its direction, occasionally produces for the time systolic murmur at the base. I have observed this not only in hysterical females, who, though of florid countenance, might, it is true, have been slightly spanæmic, but in males with palpitating heart. If the organ be the subject of dilated hypertrophy, palpitation is sometimes, but certainly not always, attended with the same murmur. In hypertrophy with dilatation there is possibly, we have seen, another dynamic source of systolic murmur: from the altered form of the cavities of the ventricles, their contained blood is probably propelled against the edges of the arterial orifices, instead of directly through them; and this misdirection of the current may, very possibly, generate murmur.

The heart may, probably, also undergo dynamic changes interfering with the closure of its valves, and giving rise to murmurs of the regurgitant class. A systolic murmur at the left apex is occasionally audible in chorea, and probably also in other neurotic affections, disappearing as the disease disappears,—which cannot be referred to inflammation or organic change of the mitral valve; which has not the usual accompaniments of a hæmic murmur; but which does seem plausibly ascribable to disordered action of the muscular apparatus connected with the valve.* Its intensity varies with different beats of the heart; its quality similarly changes; and it may even temporarily disappear. The same kind of disorder may conceivably be the cause of certain mitral regurgitant murmurs, attending dilated hypertrophy of the left ventricle, and disappearing under treatment.† Is it possible, too, that the innervation of the auriculo-ventricular valves may be disordered, so as to impede their closure? they are provided with sympathetic filaments; why may they not palpitate, as well as the aorta?

443. To return to endocardial murmurs in general: In

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* If the heart be removed from the body, an auricle cut away, the artery of the same side tied, and the cavity of the ventricle filled with fluid; and if then, a stream of water be directed upon the auriculo-ventricular valve, this rapidly closes. From this experiment of Baumgarten, the inference has been drawn that the auriculo-ventricular valves are closed by the systole of the auricles, prior to the systole of the ventricles, and that the closure is not in anywise influenced by the musculi papillares, but is much facilitated by the specific lightness of the valves themselves. The multitude of points in which the experiment fails to imitate the natural state of things in the living and contracting heart, utterly invalidates, in my opinion, the conclusion it has been forced to furnish. But, even granting that this inference be sound in *physiology*, the additional assumption of M. Hamernjk, that *morbid* conditions of the muscular structure of the heart can have no effect in preventing closure of the valves, is a palpable *non sequitur*. What! suppose that (*inter alia similia*) the papillary muscles are shortened, puckered, dwindled in muscular texture, and infiltrated with pseudo-fibrous induration-matter (as they occasionally are), will not this state somewhat, at least, interfere with the function of the valves? We shall next be told that shortening and thickening of the chordæ tendineæ produces not the least embarrassment in the play of the valves.

† Bonsey, U. C. H., Males, Oct. 1850.

seeking the causes and seat of any given endocardial murmur, the essential points in the inquiry are its relationship to the systole or diastole, and the spot of its maximum intensity on the surface of the chest. Subsidiary conditions of great importance are the direction of transmission, duration, clinical progress, quality, and pitch of the murmur; and, in addition to its own characters, the state of the heart's natural sounds, and the presence or absence of certain audible phenomena in the arteries and veins, or both, are valuable guides.

444. Each orifice of the heart may be the seat of two murmurs, constrictive and regurgitant,—with, or against, the current: the total number of cardiac murmurs connected with the orifices, therefore, reaches eight. The essential characters of these may be briefly set down as follows:—

445. *a.* A *systolic* murmur of maximum force at, and immediately above, or to the outside of *the left apex*,—but faintly audible, or wholly inaudible, at the right apex (say, the ensiform cartilage), the mid-sternal base,* and the pulmonary and aortic cartilages,—more or less clearly audible about and within the inferior angle of the scapula, and beside the dorsal vertebræ from the sixth to the ninth, audible or not round the lateral base of the chest from the cardiac to the scapular region, is essentially characteristic of *regurgitation through the mitral orifice* at the moment of the ventricular systole. This regurgitation may be the result of inefficiency of the valve, produced by chronic changes of structure (its common cause),—or by enlargement of the orifice without coeval growth of the valve (a *very* rare cause),—or occur from non-closure in acute endocarditis, in consequence of roughness of the edges of the valves and shortening of the chordæ tendineæ,—or, perhaps [442],

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* If the stethoscope be carried gradually upwards and inwards from the maximum point towards the third cartilage, it will be found that at a certain line, defined with singular sharpness, the characters of the murmur completely change: in intensity, it falls to a third; in roughness, loses greatly; in pitch, falls.

from disordered action of the columnæ carneæ. But it is not absolutely pathognomonic of such regurgitation; for, in some very rare cases, aneurism of the heart,—fibrinous coagula amid the columnæ carneæ, near the valvular portion of the ventricle, —and vegetations on the ventricular surface of the valve, have produced a murmur of this rhythm and site. The dynamic or organic nature of the murmur cannot be determined from its audibleness, or the reverse, round the lateral base of the chest.

Of very variable quality, this murmur is rarely of high pitch, generally oscillating between whispered *who* and *rr;* but I have known it sharply whistling, as of the wind through a key-hole. Once established, it is, as a rule, permanent; but when, probably, dynamic, as in chorea, it may wholly disappear. If the cause of non-occlusion act intermittently, the murmur will be present at some moments, absent at others. This I once observed in an adult male, in whom a body about the size of a large pea was suspended by a thread-like peduncle from the larger division of the valve, in such manner that it might or might not, according to accidental circumstances, have fallen within the orifice, and impeded its closure.

A mitral regurgitant murmur may completely or partially cover the first sound at the left apex. This first sound may have its natural characters in perfection at the base and at the tricuspid apex; but when intense the murmur may partially obscure the sound in both these places by conduction.

In the healthy state, the second sound is more strongly accentuated in the aorta than in the pulmonary artery. The reverse, according to Skoda, holds in cases, among others, of mitral regurgitation; and the peculiarity is explicable thus:— With every systole some blood is forced into the left auricle; that auricle, the pulmonary veins, and pulmonary artery quickly become overstretched, and the right heart requires greater effort to force the blood into the over-filled vessels; the pulmonary artery consequently presses with increased force on its column of blood, and hence intensifies its own portion of the second sound. And, as a corollary, he holds that the absence or presence of this intensification will distinguish systolic murmur at the left apex, caused by regurgitation, from that caused by friction of the blood against roughnesses in the ventricle. I do not believe, although it certainly does exist in some cases, that any such implicit confidence can be placed in this sign. I have known it wanting in cases of mitral regurgitation, even when there was no obvious tricuspid regurgitation to afford a plausible explanation, through diminished current, of its deficiency. It seems to me sometimes, too, only a pseudo-accentuation of the pulmonary second sound, from real weakening of the aortic second sound through the lessened current and diminished calibre of that vessel, that follow on long-continued mitral regurgitation. Besides, I have not found thickening or enlargement of the pulmonary artery in such connection with mitral regurgitation, as ought to obtain, were the theory described wholly accurate,—the more so, as thickening and enlargement of the left auricle really do exist in a fair proportion of these cases.

A murmur with the characters now described is the most common of the organic class,—is rarely of functional, and never, so far as I have seen, of purely hæmic origin; and is always connected with the mitral valve, orifice, or neighbouring portion of the left ventricle.

446. β. A *systolic murmur* of maximum force *immediately above, or at the ensiform cartilage*, inaudible, or nearly so, at the left apex, and, according to my experience, imperceptible in the left vertebral groove opposite the lower angle of the scapula, originates in the *right ventricle.* In the great majority of cases arising from *tricuspid regurgitation*, it may by possibility depend on sharp collision of blood among thickened and roughened chordæ tendineæ. In the former case, distension and pulsation of the auricle, vena cava, innominate and jugular veins is habitually present, that of the latter *visible;* but the absence of

such pulsation does not prove that the murmur under consideration is independent of regurgitation.

This tricuspid murmur is generally soft, and of low pitch, rarely masks the systolic sound completely, is of rarer occurrence than tricuspid regurgitation itself, and is probably not always detected when it exists. It is, absolutely speaking, rare, because regurgitation often occurs from insufficiency, without morbid change, of the valves, and because the back current is often not forcible enough for the production of a murmur. On the other hand, it escapes detection, because it is often covered by a powerful mitral murmur, and in some cases impaired in distinctness by deep-seated venous hum. In rare instances, where a mitral and tricuspid murmur coexist, a spot may be found midway between their maxima points, where there is comparatively little murmur.

In certain cases of mitral regurgitation, the attendant murmur may be so deadened at the left apex by emphysematous lung, as to be better audible at the right apex, and so simulate a tricuspid murmur.*

447. γ. A *systolic murmur*, of maximum force, *at mid-sternum, opposite the third interspace* (or, it may be, the upper part of the fourth rib), abruptly losing force between this point and the left apex, where it may be almost inaudible, faintly perceptible at the second left cartilage, clearly audible at the second right cartilage, the notch of the sternum, and the left vertebral groove, opposite the second, third, and fourth vertebræ, thence rapidly losing strength downwards, and disappearing about the sixth, originates at the *aortic orifice*. It habitually signifies constriction, smooth or rough, of that orifice, and in rare cases has been traced to fibrinous coagula impeding the egress of the blood. But cardiac murmur, very closely simulating this type, may be produced in cyanosis and in certain aneurisms of the heart and of the aorta. This

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* Hutchinson, U. C. H., Males, vol. viii., p. 335.

murmur is commonly of high pitch, loud, prolonged, and harsh; an hypertrophous ventricle concurring, increases its intensity, and may give it a drawling prolonged character, if the sides of the orifice be much contracted.

This is, besides, as already explained, the commonest of intracardiac hæmic murmurs.

Although audible at the aortic cartilage, it is distinctly fainter there than at the base; if as marked, and *à fortiori* if more marked there than opposite the valves themselves, disease of the arch of the aorta itself adds to the murmur.* The same inference is justified by any notable difference in pitch in the two spots.

448. δ. A *systolic murmur*, of maximum force *at the sternal edge of the third left cartilage*, or a little lower down, audible at the pulmonary cartilage, almost inaudible at the aortic cartilage and at the apex, and imperceptible in the back, indicates *obstruction at the orifice of the pulmonary artery*, simple roughness in its valves, or, as noticed by Dr. Elliotson, pressure on the vessel by adventitious masses in the pericardium. All these causes combined, however, are so unusual, that few persons have actually met with such a murmur: some simulating murmurs will be described hereafter. I have only observed one of the kind; and as, in this instance, there was no *post-mortem* examination, its site cannot be held to have been certain, seeing that we have no established experience of its characters.

449. ε. *A diastolic murmur*, of maximum force, *immediately above and about the left apex*, and conducted in the same directions, though less extensively, as systolic murmur of the same seat, indicates *obstructive narrowing of the mitral orifice*, or simple roughness of the auricular surface of the mitral valve, or both states combined. Skoda affirms that murmurs of the two

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* There is one kind of exception to this statement: if the apex of the right lung be solid, the murmur may be more marked at the aortic cartilage than at the base, though the aorta is healthy. I have never known more than slight excess under these circumstances.

sources may be distinguished by the condition of the second sound in the pulmonary artery: it will be loud to excess in the case of narrowing; unaffected in that of simple roughness, unless other causes of reinforcement be present. The distinction will hence, even if well-founded, be clinically useful only in cases where the absence of mitral regurgitation is certain.

I have never heard this murmur of great intensity, nor high pitch; it is, however, sometimes prolonged. It is rarely loud enough to cover the second sound completely, even at the left apex.

This murmur is commonly spoken of as diastolic in rhythm; but in point of fact it is rather post-diastolic or præ-systolic, than precisely coincident with the diastole.

This murmur is not very unfrequently wanting, where constriction is found after death. Sometimes the deficiency may be fairly referred to the weakness of the auricular systole and smoothness of the constricted orifice;* where the constriction is slight, the friction will also be but slight. When deficient, as it has positively been, in cases of marked contraction, Hope thought the deficiency depended on the very fact of the extreme smallness,—an explanation not over plausible.

I have known this murmur come and go from day to day in a case where the mitral orifice was very greatly contracted and rigid:† probably from varying force of the heart's action.

450. ζ. *A diastolic murmur*, of maximum force, at *the ensiform cartilage*, most faintly audible at the left apex, and inaudible at the base, would probably indicate *tricuspid narrowing*, were there a hypertrophous auricle behind that orifice,

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* In the case of Denham (U. C. H. Males, vol. vi. p. 77), the long tongue of the mitral valve was much thickened; at its union with the other tongue it was puckered; and a mass of ossiform substance, as large as a very small marble, smooth on the auricular, rough on the ventricular surface, had formed at the union of the two tongues. Here there was no mitral constrictive murmur.

† Kernis, U. C. H., Females, vol. ii. p. 240.

to give force to the current. The little I know of the murmur by experience will be found in the description of Diseases of the Orifices, in Part II.

451. η. A *diastolic murmur*, of maximum force *at mid-sternum, opposite the third interspace or fourth cartilage*, conducted (with some exceptions, to be mentioned presently) on the same principle as the systolic murmur of the same site, indicates *regurgitation through the aortic orifice.*

This murmur may be heard with almost as much intensity about the ensiform cartilage as opposite the third interspace—in this point of view differing materially from constrictive aortic murmur: the more distant conveyance downwards in the former case probably depends on the downward direction of the current producing the murmur. So marked is the fact, that unless with care the murmur of aortic regurgitation might be mistaken for one of tricuspid constriction. I have even known the murmur louder at the upper edge of the xyphoid cartilage than at the base of the heart.* It is remarkable that the conduction is more perfect towards the right than the left apex,—consequently not, as might have been supposed, in the direction of the communicating ventricle: concerning this point I find my experience at variance with the statement of Hope. The murmur may, though well marked in front, be quite inaudible in the vertebral groove.*

The second sound of the heart may be covered completely at the maximum point of the murmur; or it may be heard at the beginning of, during, or at the close of this. In the first case, the valves are utterly incompetent: in the varieties of the second case, one division of the valve may flap naturally, or all three imperfectly, and so produce an imperfect second sound; or the second sound heard may be wholly that of the pulmonary valves.

The most common of these compound conditions of diastolic murmur and sound at the base, is that of murmur abruptly brought to a close by sound; a state pretty accurately rendered by the whispered symbols *phwi...tt* or *phwe...tt*,—the *tt* being sharply accentuated. Skoda thinks, under these circumstances, the murmur is due to friction of the recoiling blood during the arterial systole against the roughened coats of the aorta, and the sound to the natural closure of the valves without regurgitation. This explanation may hold in some instances; I am certain it will not apply to all. For I have observed this state of basic diastolic sound, where the existence of marked visibleness of all the superficial pulses left no doubt in my mind of the existence of aortic regurgitation,—the patients being besides young and free from notable hypertrophy of the left ventricle [482].

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* Flood, U. C. H., Males, vol. vii., pp. 265, 266.

Unless the murmur be of very great intensity, the second sound may be well heard at the left apex,—it may even be strongly accentuated there.[*] The causes of this have already [403] been inquired into.

Aortic regurgitant murmur is usually of aspired blowing quality, sometimes almost hissing, generally of low pitch, rarely rough, weak as a rule (though I have known it extremely loud, and it is said to have been heard at a little distance from the surface), and of considerable duration, habitually filling the post-diastolic silence.

The causes of this regurgitation are the ordinary ones entailing incapacity of valves; I have known sudden rupture of one of the sygmoid valves produce it.[†] "Reticulation" of the valves carried to extremes, a conceivable source of murmur, has never fallen under my notice as its cause;[‡] but perforative destruction on a large scale will of course do so. Mere insufficiency of valves, healthy in themselves, but too small to fill the morbidly widened mouth of the aorta, has in rare instances produced this murmur. In exceptional cases it is developed during the *acute* period of endocarditis.

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[*] Flood, U. C. H., Males, vol. vii., p. 265.

[†] Gordelier, Consumption Hospital, Males, vol. i.

[‡] Probably because reticulation affects that part of the valves — the neighbourhood of their free border—which naturally takes no direct part in preventing regurgitation. If, as is taught by some persons, reticulation of the sygmoid valves were a real cause of regurgitation, pulmonary diastolic murmur would be common, seeing that reticulation of the pulmonary, is very nearly as frequent as of the aortic, valves.

452. θ. From experiments on animals, it appears that a *diastolic* soft prolonged murmur, audible down the ventricle, may be produced by artificially rendering the *pulmonary valves insufficient.* But this regurgitant murmur is of excessive rarity in man: I have known an aortic regurgitant murmur more distinct at the second left than the second right cartilage—an obvious source of fallacy.

453. In respect of relative frequency, I should be disposed to place intra-cardiac murmurs of *organic* origin in the following order, commencing with the most common:—mitral regurgitant; aortic constrictive; aortic regurgitant; mitral constrictive; tricuspid regurgitant; pulmonary constrictive; pulmonary regurgitant; tricuspid constrictive.

454. These murmurs may be variously associated; the following combinations are arranged in the order of their relative frequency:—aortic constrictive and mitral regurgitant; aortic constrictive and regurgitant; mitral regurgitant and aortic regurgitant; mitral regurgitant, aortic constrictive and regurgitant; mitral regurgitant and obstructive; mitral regurgitant and tricuspid regurgitant; mitral regurgitant and constrictive, aortic constrictive and tricuspid regurgitant; tricuspid constrictive and mitral constrictive.*

455. No one organic murmur involves, as matter of necessity, the presence of another; a direct murmur may exist at any valve, and an indirect be absent, and *vice versâ.*

456. When two murmurs co-exist at the same orifice, they are readily distinguishable by their rhythm, their quality, their pitch, and by their expiring or aspiring character. I have

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* The anatomical conditions of this singular combination are exemplified by Carswell's Drawings, U. C. Museum, Portfolio A, No. 285.

great difficulty in believing, with Skoda, that mitral systolic and diastolic murmurs, forming one quasi-continuous noise, shall be in nowise distinguishable from a single murmur, until slackened action of the heart distinctly separates them into two.

PERICARDIAL MURMURS.

457. Pericardial murmurs are divisible, in respect of quality and of the mechanism of their production, into four chief species: (*a*) Friction or attrition-murmurs; (*b*) continuous murmurs; (*c*) clicking murmurs; (*d*) murmurs produced by bending of layers of exudation-matter. Roughness of surface is the essential statical condition of them all,—movement their essential dynamic element.

458. (*a*) Attrition-murmurs, all of them, more or less, distinctly suggestive of rubbing of surfaces of variable character against each other, occur in a greater number of varieties even than pleural friction sounds. They resemble, for instance, *grazing*, *coarsely rubbing*, *grating*, *scratching*, *creaking*, *squeaking*, *and prolonged whistling* noises. Traceable as all these varieties commonly are to collision of surfaces roughened with lymph, the interest attached to their distinction must turn mainly on their being severally connected with some particular state of that lymph. Now, experience does not show any such necessary connection; though it be true, for the most part, that at the very earliest period the murmur is of grazing quality, like the sound produced by rubbing pieces of silk together, and that as the exudation hardens, and gathers into irregular peaked elevations, the quality becomes coarsely rubbing, grating, creaking, this sequence of changes is subject to constant exceptions. Creaking friction-sound, so loud as to be audible *three inches from the end of the stethoscope applied to the surface in the ordinary way*, may depend, I know from actual observation, on tough exudation-matter with fine rough elevations; and I have noticed distinct, though slight, creaking quality, when the exudation-matter was found to be of almost creamy softness.

Dr. Taylor,* too, has known signs of moderate liquid effusion coexist with friction-sound of this quality,—a fact showing that peculiarly rough attrition is not required for its production.

459. (*b*) When exudation-matter and fluid coexist, the former imprisoning the latter in its meshes, the heart's action produces a peculiar continuous rumbling, or squashy churning sound, just such as, we find, occasionally occurs in the pleura. This is rare, but, once heard, can never be forgotten.

460. (*c*) Occasionally sounds are heard of peculiar clicking character, one or two, with each beat of the heart, which are only distinguishable, at the time, from modifications of the valvular sounds, by their non-synchronism with these, and by the extreme irregularity of their occurrence. I have satisfactorily traced these clicks to the pericardium, and further, in all probability, to the separation, without attrition, of surfaces glued together with exudation-matter.†

461. (*d*) It has appeared to me that sound is sometimes generated in layers of firm false membrane, though so perfectly agglutinated together that attrition or separation of the apposed surfaces is physically impossible. The quality under such circumstances is probably variable: in the only positive instance of the kind which I have observed, it was faintly creaking. The bending and crumpling of tough false membrane may conceivably generate such sound.

462. The varieties of pericardial murmur may be heard over all parts of the cardiac region from the roots of the large vessels to the apex. But I have never detected the churning variety except about the apex, nor the clicking variety except in the site of the large vessels. The finest shades of grazing sound are most common behind the sternum. Different qualities of murmur may be heard at one and the same time in some cases

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* Brit. and For. Med. Rev., vol. xxiv.

† Barr, U. C. H., Males, vol. vii., p. 328-9. Here there was clicking pericarditic murmur at the second left cartilage and interspace; while the apex was raised, and the præcordial region bulged by fluid.

over different parts of the heart; but this is not common. As a rule, friction-sound is most clearly and frequently detected below the third interspace, probably simply because, below that part, there is little lung intervening between the pericardium and surface. Limited in some cases to a mere point of surface, it may reach from the clavicles to the epigastrium and from nipple to nipple.* In rare instances, friction-sound in the pericardium is audible in the back, between the scapulæ and the spine,—I mean in cases where mere intensity of sound cannot be held to explain the fact. It has been found single in the back, too, while double in front. It seems sufficiently likely that in some, at least, of the cases where it is alleged to have been confined altogether to the back, the friction heard may have been produced in an inflamed pleura by the cardiac impulse. Pericardial friction is usually abruptly limited.

463. Pericardial murmurs vary exceedingly in intensity; so delicate in some instances, especially when of grazing or clicking quality, that the closest attention is required for their detection; in other cases they may be heard in the posterior and lateral regions of the chest, and even, as already mentioned, at a considerable distance from the surface in the præcordial region. On the whole, their intensity is greater than that of pleural-friction sounds—a fact accounted for by the comparative abruptness and energy of the motion inducing them.

464. When the entire cardiac surface is the seat of friction-murmurs, the maximum amount of sound exists, according to some writers, about the nipple; to others, behind the sternum. I am satisfied no rule of the kind can be established: I have known the point of maximum intensity change from the fourth interspace to the fifth, thence to the ensiform cartilage, and thence to the nipple, within twenty-four hours.† Neither can

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* Case of Mr. S., seen with Dr. J. H. Davis.

† Campion, U. C. H., Females, vol. vi., p. 260. Here, at one time, it was loudest at the base.

any particular part of the cardiac surface be fixed on as the absolute seat of loudest friction-sound; it is sometimes, though rarely, excessively loud even about the base.

465. Pericardial friction may accompany both the systole and diastole, or either singly; its co-existence with the systole alone is not rare; with the diastole alone, infrequent. When of regular rhythm, the friction-murmur falls a little after the corresponding valvular sound. But such regularity as this is the exception, not the rule; the friction-sound may be very distinct during the post-diastolic silence. Friction co-existent with the systole is generally, but by no means constantly, sharper and louder than with the diastole.

466. Pericardial murmurs are so deficient in tone, that their pitch can scarcely be estimated even rudely, although they embrace in their different varieties a considerable compass of sounds. And, indeed, no practical hint, that I know of, is derived from their pitch; except that, generally speaking, the higher this is, the drier and rougher the material of attrition. The pitch of a pericardial murmur may sometimes be raised by pressure with the solid stethoscope, and also by full inspiration.*

467. Pericardial murmurs are, as a rule, distinguished by their superficial character: they appear to be produced immediately underneath the integuments. But if the physical cause of the murmur be placed beyond the limits of the triangular portion of the heart, uncovered by lung, they lose this character, and seem of more or less deep origin. This is sometimes particularly observable about the large vessels, before any effusion has occurred to push the edges of the lungs aside; their apparent depth then contributes to assimilate them in certain varieties to valvular murmurs.†

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* Bingley, U. C. H., Males, vol. vii., p. 114.

† Skoda attempts to show that the clinical distinction of sounds, produced deeply and superficially, is a delusion. If a stethoscope be placed on the chest of a dead body, the sounds produced by tapping different parts of the inner surface of the parietes, near and distant, will, as far as I have been able

468. Various circumstances modify the intensity and superficial extent of these murmurs,—the physical condition of the pericardium itself remaining the same. The most important of these is the energy of the heart's action: the greater this, the louder the friction-sound. The weakness immediately ensuing on blood-letting, approaching syncope, the action of digitalis and aconite, all lower its intensity: if successive systoles be of very unequal force, friction, absent with one, is present with another, impulse. An hypertrophous and dilated heart gives, *cæteris paribus*, the maximum quantum of attrition-murmur. Attrition-murmurs are more marked in expiration than in inspiration; and when the trunk is bent forwards, than in the recumbent position. Moderate pressure, especially with the solid stethoscope, commonly intensifies them; strong pressure may mask them completely.

469. Pericardial friction may appear very rapidly after the cause of inflammation has acted. Thus, in the remarkable case at University College Hospital where fatal perforation of the œsophagus and pericardium was produced in the attempt to swallow a sword, friction was detected by my then Clinical Assistant, Mr. Tidmas, thirty minutes after the accident.

470. The duration of friction-murmur varies very greatly. I have known it appear and disappear, finally, within six hours, —of the grazing variety, it is true, but still of unmistakable characters. In illustration of the other extreme, I may refer to a case in which it continued audible for upwards of three months, especially at the lower part of the sternum.*

The total disappearance of pericardial murmur may, if it be

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to ascertain, differ very sensibly: exceptions occur, it is true; but these do not destroy the rule.

* Kennedy, U. C. H., Males, vol. i., p. 67. First heard on the 27th Oct., it was last heard on the 5th Feb. following, long after the man's discharge from the hospital, and apparent restoration to health. How much longer it may have continued I know not, as the man was not afterwards examined, though often seen again on duty in the streets as a policeman—a fact showing the completeness of his recovery.

slight in amount, almost immediately follow general or local bleeding; this disappearance may be only transitory, however, from weak action of the heart: or a pericardial murmur may abruptly disappear from rapid agglutination of the entire of the affected surfaces. I have known it impossible to find a vestige of friction in a case where, six hours before, the *entire* cardiac region was the seat of intense rubbing sound. But, generally speaking, the progress of agglutination is materially slower than this, and for several days one or more limited spots may be found, where the collision continues soniferous (sometimes in the churning variety), after the adhering process has commenced. The sudden cessation of friction-sound, through absorption of exudation-matter, is less easily conceived; and where such cessation occurs, independently either of agglutination, or of rapid pouring out of effusion, the inflammation had probably produced very little indeed of that matter.

471. In the majority of cases where friction abruptly disappears, the change depends on fluid effusion, separating the pericardial surfaces from each other: it is consequently oftener an evil, than a good, sign. With collateral circumstances, such as the size of the heart, conformation of the chest, absence or presence of adjoining pleural adhesions, and the accident of partial soft adhesions having, or not having, occurred, before the fluid begins to accumulate, the amount of liquid required to annul friction-sound varies. A case of Dr. Taylor's (loc. cit.) shows that friction may continue when eight ounces of fluid, or thereabouts, have accumulated: but generally less removes it. Disappearing with the occurrence of effusion, friction-sound pretty frequently returns when the fluid is absorbed: this returning, or redux, friction generally, but not always, appears first about the great vessels and base. Pleuritic friction, we have seen, is more commonly caught at the redux period; pericardial, certainly, at the outset of the inflammation. The second disappearance of friction may be abrupt, slow and gradual, irregular with recurrences, or rapid over the heart

generally; while in a single spot or two, most commonly either at the apex or about the great vessels, some form of the murmur remains for a time.

472. Friction in the pericardium, the cause of which lies within itself, signifies inflammation of the membrane. It is exceedingly probable that mere dryness of the surfaces will suffice to produce the grazing variety. I know from observation that mere vascularity of a very small surface, without a particle of lymph, may produce faint rubbing noise;* but exudation-matter is its common physical element. In the great majority of cases, exudation-matter forms on both serous surfaces. Dr. Taylor relates a case where, the cardiac surface alone being affected, ordinary friction was almost completely absent: still, however, it appears to have existed, single and systolic, to a slight amount about the base. If exudation exist on the posterior aspect of the sac only, friction will commonly be inaudible.

A former attack of pericarditis will or will not prevent the development of friction-sound with a new attack, according to the state in which it has left the serous membrane and cavity of the sac. If it have left a state of perfect agglutination behind it, then, unquestionably, friction is impossible; if of loose adhesions, new lymph may be thrown out between these, and friction will be developed.† But, probably, old pericarditis will always have a tendency to limit the extent and regulate the site of new friction-sound.

Calcification of exudation-matter within the pericardium, and also tuberculous and carcinomatous disease, may be conceived to produce permanent friction sound; but I have not observed this.* Whether fibrinous, cancerous, or other matter within, and in the walls of, the heart, may, by simply elevating the cardiac surface, produce friction-sound, independently of pericardial irritation, I do not know from experience.

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* F. Parker, U. C. H., Males, vol. iv., p. 177, Dec. 1848. A rub may attend the impulse at the apex with knocking sound, independently of any pericardial irritation, as far as can be even suspected; *e. g.*, cases of Sus. Roberts and Bonsey, U. C. H., Oct. 1850.

† Craddock, U. C. H., Males, vol. vii., p. 336, admitted for his eighth attack of rheumatic fever: for five of these he had been treated in the hospital, and had pericardial friction on every fresh seizure.

473. The distinction of pericardial from neighbouring pleural friction, turns mainly upon its difference of rhythm. But sometimes cardiac action produces friction in an inflamed pleura adjoining, the pericardium being unaffected. The distinction of this kind of friction, pleural in site, and cardiac in rhythm, is not always easy: though commonly limited to the confines of the cardiac region in front, it may be audible in the back on the left side; † its intensity is generally increased by the act of breathing,—the pleural surfaces being then in motion of one rhythm, are more easily thrown into motion of another, than if at rest; it seems sometimes limited to the time of expiration.† The following circumstances argue in favour of friction of cardiac rhythm being of pleural, and not pericardial, origin: the limitation of the sound to either edge, generally the left, of the cardiac region; fixity in one or more particular spots; cessation complete, or, what is more common, occasional with certain beats of the heart, when the breath is held; and marked unsteadiness in the intensity and quality of the friction-sound. Local dry pleurisy, close to both sides of the heart, and productive of friction both of pleural and of cardiac rhythm, is sometimes followed by dry pericarditis: the presence of the latter inflammation can scarcely be affirmed, until effusion takes place; for the distinction of the true pericarditic friction-sounds is then most difficult.‡

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* On the contrary, indeed, I have known very positive elevation of the pericardium by carcinomatous nodules unattended with the least friction sound; but the serous surfaces were smooth. Case of R. Smith, Multiple Subcutaneous Cancers, *Med. Times*, August, 1852.

† Wilkinson, U. C. H., Males, vol. ix., p. 284. But true pericardial friction may be most marked in expiration: *e. g.*, W. Price, U. C. H., Males, vol. vii., p. 214.

‡ Two cases of this kind (Hayes and F. Smith) once fell under my notice

474. Pericardial murmur is, in the great majority of cases, easily distinguished from that of endocardial origin by the following characters and circumstances: its rubbing quality; its superficial character; its abrupt limitation and non-transmission in the course of endocardial murmur; its changeableness in precise seat and intensity from hour to hour; the increase it undergoes in sharpness and extent when the patient bends forward; its occasional increase by regulated pressure of the solid stethoscope;* its want of perfect synchronism with, or fixed relationship to, the heart's sounds; and its being sometimes accompanied by friction-fremitus, which can scarcely be confounded with valvular thrill. Pericardial murmurs are commonly rougher than *acute* endocardial murmurs. Hope, indeed, held that a diastolic *rough* sound is of necessity pericardial, endocardial diastolic murmurs never possessing this character; but in this he was absolutely wrong: an aortic diastolic murmur may be very rough. On the other hand cases occur where it is next to, or absolutely, impossible to decide on the peri- or endocardial origin of a murmur. This difficulty is oftenest felt about the base and great vessels, and when the sound is of clicking character. Sounds really produced at the valves of the aorta, may not be transmitted along this vessel,—as a consequence simply of their feebleness.

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at the same time; U. C. H., Ward 4, Oct. 1850. Vide Clin. Lect., loc. cit. p. 389.

* I leave this clause as it stood in the first edition, although, I confess, I daily lose faith in the efficacy of pressure as a positive means of distinguishing intra-cardiac and pericardial murmurs. There are several reasons, it appears to me, why implicit trust cannot be placed in the effects of pressure. First, an indubitable friction-sound may be weakened by pressure, if carried beyond a certain amount; secondly, a pericardial rubbing-sound may be sometimes induced by pressure, where the pericardium is free both from exudation-matter and vascularity; thirdly, a true endocardial murmur may be increased in thin persons with flexible chests by regulated pressure,—possibly not endocardial murmurs of all sites and all rhythms, but certainly systolic basic murmur. (*E. g.* Lewis, U. C. H., Females, vol. ix., p. 320; Gay, U. C. H., Females, vol. ix., p. 316.) The increase in intensity seems to be sometimes merely the effect of improved conduction.

475. The action of the heart sometimes produces rhonchal sounds in the adjacent lung,—cavernous, and coarsely or finely bubbling. The rhythm of the rhonchi, and their persistence during suspension of the breath, disclose their mechanism.

476. A double rasping friction-sound, so loud as to be audible at two inches' distance from the chest, existed in Dr. Swett's remarkable case of enlarged granular liver, where the pericardium and valves were perfectly natural.

RESPIRATORY MURMURS IN THE CARDIAC REGION.

477. In the natural state the respiration is clearly audible over the entire cardiac region, though somewhat enfeebled about its centre. Enlargement of the heart widens the superficial extent of this feebleness; pericardial effusion does so also, and still more in proportion to the percussion—dulness existing,—to so sensible an amount, indeed, that the excess is clinically useful *as an aid* in distinguishing these two states of disease under difficult circumstances.

Pericardial adhesions, especially if attended with close union of the pericardium to the ribs, and agglutination of the adjoining pleural surfaces, will maintain the natural amount of audible respiration in the pericardial region, through the course of a subsequent attack of pericarditis with liquid effusion. Under these circumstances, persistence of respiration in front of the heart would, as I have elsewhere shown, be a sign of pericardial adhesions.

VOCAL RESONANCE IN THE CARDIAC REGION.

478. In the state of health, vocal resonance is weak, or actually null, in the præcordial region. In some cases of pericardial effusion, the resonance is not only morbidly loud on the confines of the liquid, but acquires an ægophonic quality. In the only instance in which I have actually observed this, the state of the lung adjoining the distended sac accounted for the increase of resonance,—the fluid simply gave this an ægophonic quality.*

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* Harley, Consumption Hospital, Males, vol. i., p. 76.

GREAT ARTERIES.

SECTION I.—INSPECTION.

479. The outline and movements of the large arterial trunks are not visible to the eye in the state of perfect health. In very emaciated persons the pulsation of the aorta in the epigastrium may be seen, however; and, if the arch of that vessel lies unusually high, slight impulsive motion may be visible in thin people above the sternal notch. The carotid pulses can scarcely be seen, if the neck be tolerably provided with soft parts: the same statement applies to the smaller arteries. No expansile character can be detected in any of these motions.

480. With these exceptions, notable visibleness of arterial pulsation, especially with onward locomotion of the artery, is an unnatural condition, depending on (*a*) general or local excitement of the circulation; (*b*) special disease of the heart; or (*c*) disease of the visible vessel itself.

481. (*a*) General excitement of the circulation, it is well known, produces visible throbbing action in the innominate artery and carotids, which passes away with that excitement. The vessels, however, simply pulsate, and undergo no onward locomotion. Visible pulsation of the epigastric aorta may be similarly induced. The arteries leading to an inflamed part often beat visibly.

482. (*b*) Aortic regurgitant disease, as first shown by Dr. Corrigan, renders the pulsation of the superficial vessels visible: the vessel moves forward too, in the direction of its axis,—sometimes vermicularly. The radial arteries at the wrist, the temporals, and the posterior tibials behind the malleoli are the

usual seats of the appearance; but in highly marked cases the carotid, brachial, femoral, and external iliac arteries distinctly present it. To what extent may this sign be trusted to, as evidence of aortic regurgitation? In the first place, no well-marked case of that disease has ever fallen under my notice in which visibleness in the superficial pulses was not more or less obviously present. In the second place, I have never observed *highly marked and extensive* visible pulsation without aortic regurgitant disease. But in the third place, in aged persons of thin frame, whose vessels are calcified and tortuous, a slight degree of the phenomenon may certainly be noticed in the smaller arteries, independently of any affection of the aortic valves; and if the left ventricle be hypertrophous in such persons, the amount of visibleness will be materially greater; but, still, will fail to affect the larger trunks. I have never known simple or eccentric hypertrophy alone produce this sign in persons under the age of forty, whose limbs were even moderately well covered with flesh: it is affirmed that the peculiarity has occasionally been traced to this cause solely, but the qualification regarding age, my own observation leads me to make, is not referred to. Hope thought that where there was mitral regurgitation or contraction to any amount, aortic regurgitation failed to render the pulse visible. This seems somewhat theoretical, and is positively opposed to what I have seen.*

483. (*c*) The impulse of arteries locally diseased is much better studied by the touch than by the sight.

SECTION II.—APPLICATION OF THE HAND.

484. The systole of the ventricles conveys to the entire extent of the arterial system an undulatory and somewhat expansile motion, easily felt in vessels, of a certain size, lying

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* Denham (U. C. H., Males, vol. vi., pp. 69—78), had marked mitral regurgitation and constriction associated with aortic regurgitation, yet his radial, brachial, posterior tibial and femoral pulsations were well visible.

within reach of the fingers, and known as the arterial pulse or diastole. Each arterial diastole is followed by a contraction or systole. In vessels close to the heart the pulse is perfectly synchronous with the ventricular systole, but falls more and more behind it, in point of time, the further the artery from the central organ. With this qualification, the synchronism of the pulse and ventricular systole is perfect in health; the altered rhythm of the latter, produced by changes of posture, and all other physiological causes, is impressed on the former. In diseases of the heart, various perversions of this synchronism occur, and have been already described. The systole of the arteries, synchronous with the heart's diastole, is pulseless.

485. The characters of the pulse which may have diagnostic signification in cardiac diseases are as follows:—the diastole may be quick or slow, short or prolonged, soft or hard, loose or tense, empty or full, small or large, equal or unequal in force, and other characters, in successive beats; and the rhythm may vary in the different forms elsewhere noted [361]. The stroke may be vibratory, jerking, undulatory, bounding, or reduplicate (bis-feriens): these terms are in themselves sufficiently explanatory of the states to which they refer.

486. The pulsation of the thoracic aorta can be felt only in one spot in health,—above the sternal notch. If the finger be pressed downwards in that position, the patient's head being at the same time bent forwards, slight impulse is, in the majority of cases, detected: where the arch lies high, there will, of course, be very distinct movement. True aortic impulse is felt directly in the middle line; impulse inclined to the right side originates in the innominate artery. The pulsations of the abdominal aorta may be felt, if pressure, proportional to the thickness of the parietes and intervening viscera, be made with the hand or stethoscope. The movement is simply one of elevation of variable force. Except in very thin persons, it is impossible to feel the beat of the vessel laterally.

487. In the various forms of dilatation of the arteries, the

dilated portion pulsates with undue force, unless it be more or less completely filled with solidified fibrine. The character of the movement becomes *hammering*,—its force sufficient, in some cases, to shake the entire trunk and limbs. Besides, the diseased vessel becomes, under certain circumstances, the seat of a double impulse with each contraction of the left ventricle; the arterial systole conveys to the hand an appreciable shock. The systolic pulsation is expansile, as well as heaving, in character; but the distinction of the former quality of impulse is not always practically easy. Now, in the case of the thoracic aorta, there is no artifice by which obscure expansile movement may be rendered distinct: in that of the abdominal portion of the vessel, the hands may be sometimes slipped deeply on either side of the vessel, and a double sideward impulsion sometimes detected, which escapes discovery when the examination is made in front only. Still, it must be confessed, the pulsation of the healthy aorta, when pushed forwards by an enlarged vertebra or tumour, cannot always be distinguished by this plan from that of enlargement of the vessel itself.

488. Arterial pulsation is in various forms of disease accompanied with thrill, occasionally more intense than that of valvular origin. Simple peripheric dilatation of a vessel, especially if it be roughly calcified, is a more efficient cause, than sacculating aneurism, of this phenomenon; and a spanæmic state of the blood contributes greatly to intensify it, when other conditions are favourable. Thrill may, in such a combination of circumstances, be felt two or three inches beyond the limits of the dilated vessel. Certain morbid states of the blood, especially spanæmia, will suffice, independently of any textural disease, to produce arterial thrill, which, though slighter in amount, is generally more diffused than that of organic origin.

SECTION III.—PERCUSSION.

489. In the natural state, the thoracic aorta has no appreciable influence on the percussion-sound of the surface, beneath

which it lies. Even when the arch lies high, the resonance at the sternal notch can scarcely be said, practically speaking, to be affected.

490. But when the vessel is notably dilated, dulness under percussion, of course, occurs, proportional in extent and intensity to the amount of enlargement,—modified in some degree by the fluid or solid state of the contents of the dilated spot, and, to a serious degree, by the vicinity of the enlargement to the surface, and the condition of the intervening textures.

The dulness of a thoracic sacculated aneurism is not coextensive with its dimensions. From its more or less globular form, a limited portion only of the sac reaches the chest-walls, and the observer cannot safely percuss with sufficient force to detect its deeply-seated parts. Hence, practically, an intra-thoracic, and even an abdominal, aneurismal sac, is always larger than the results of percussion would indicate. In non-saccular dilatation of the aorta, percussion is, for obvious reasons, a surer guide to the size of the enlargement.

The resistance of a sac filled with fibrine is highly marked; and the deficiency of elasticity so peculiar, as to help in distinguishing the dulness under percussion from that of other states; as, for instance, tuberculisation of adjoining lung. The line of union of simply condensed lung and a saccular dilatation is, however, with much difficulty to be drawn, even with the help of this peculiarity.

It is difficult to fix the smallest amount of dilatation of the arch of the aorta that can be clinically demonstrated. Much will depend on the pains taken in the investigation,—much on the thinness of the chest-walls and the healthiness of other parts; but much more on the precise site of the dilatation. In a case where the dilatation increased the width of the vessel, when cut open, by two inches, and hence increased the diameter of the unopened vessel by only about two-thirds of an inch, the fact of its existence, suspected from thrill and undue impulse, was *proved* by percussion. But here the right angle of the arch, the point where the vessel nears the surface most closely, was affected. So small an amount of dilatation could not have been detected in any other part of the arch.* Physical evidence will always be difficult to obtain, where a small sac is situated at the posterior surface of the vessel; but by careful percussion in the course of the arch, a sac as large as a good-sized walnut may be discovered, if it lie anywhere between the second right interspace and the left border of the sternum, and there be no special and unusual source of difficulty in the way.

SECTION IV.—AUSCULTATION.

491. *Arterial sounds.*—Two sounds, synchronizing with the systole and diastole of the heart, are, in the state of health, clearly audible in the course of the pulmonary artery and arch of the aorta: gradually weakening in force in the thoracic division of the latter vessel, they are with difficulty caught in the lumbar region, but by depressing the abdomen with the stethoscope, may readily be heard in front. In some persons, however, a single sound, synchronous with the systole of the heart and diastole of the vessel, can alone be detected below the chest.

In the carotid and subclavian arteries almost always, in the axillary and femoral in a fair proportion of cases, the sound continues double; in vessels more remote from the heart, a single sound only, synchronous with the arterial diastole, can be heard. The more active the circulation, the larger the vessel, the thicker its walls, provided their elasticity be not impaired, the more marked is the sound perceived on auscultation. It is not unusual to find a single, very rarely a double, sound even in the brachial and radial arteries, where these conditions are favourable.

492. In the vicinity of the heart, the arterial sounds closely

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* H. Gordelier, U. C. H., Males, vol. iii., p. 331, and Consumption Hospital, vol. i., p. 56: the patient was extremely thin, too; an accidental aid of no mean importance.

resemble the cardiac, in quality, pitch, and proportional duration; at a short distance from the heart, both become equalised in length and loudness; sometimes in the carotids the second is the longer and louder of the two. At any distance from the heart the sound loses tone completely.

If the vessels are full, and the blood of natural composition, the arterial sound is duller, but more prolonged, than when the mass of blood is small, and its quality thin. The arterial sounds are louder in females, children, and thin persons, than in males, adults, and stout people. Their intensity is increased by slight pressure; a blowing murmur takes their place, in a large proportion of persons, under stronger pressure.*

493. The arterial sounds heard in the vicinity of the heart are, doubtless, in great part transmitted from that organ: as the second can rarely be heard at any distance from the heart, the inference indeed arises that it is solely a transmission-sound, and that the systole of the arteries, at least of the minor ones, is noiseless. But that the impulsion and friction of the blood against the vessels, and vibrations of their walls, during their diastole, with the current-like motion given to their column of blood by the form of the vessels, generates sound, cannot be doubted; it is not conceivable that sound, audible in the popliteal or radial artery, is the mere result of conduction from the heart. Besides, it is not very uncommon to find the first sound in the carotid artery, oftener in the right than the left, stronger than at the aortic orifice.

494. The natural amount of arterial sound is capable of increase or diminution both in the aorta and pulmonary artery.

But the only modification of clinical significance falling under this head is increase of the second sound in the pulmonary artery,—a state connected by Skoda directly with hypertrophy or excessive systolic action of the vessel itself, indirectly with mitral constriction and regurgitation, and with hypertrophy of the right ventricle [445].

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* But it is an error to suppose that any amount of pressure will produce murmurs in all persons and in all vessels: the contrary may be hourly verified at the bed-side. The smaller the artery the more easily is murmur generated.

495. *Arterial Murmurs.*—The arteries, like the heart, become the seat of murmurs, either from organic change or independently of this.

496. *Organic* murmur in the intra-thoracic arteries varies in intensity from a scarcely perceptible sound to one audible even at a slight distance from the surface; is limited to a small portion, or, more rarely, extends over a considerable tract of the vessel; presents all the varieties of quality noted in cardiac murmurs; varies in pitch from that of the word *awe* whispered in inspiration, to a high whistling note; is of distinctly intermittent rhythm, single, either systolic or diastolic, or double; and either short and abrupt, or prolonged and slow.

497. The organic conditions of arterial murmur are referrible to (1) change of form of the vessel; (2) to a modified condition of its internal surface; (3) to both these states combined; and (4) to communication between an artery and some portion of the venous system.

498. (1) Peripheric dilatation, or simple lateral sacculation of an artery, especially if abrupt and well-marked, by altering the direction of the blood-current, generates murmur, even if the internal surface be perfectly smooth. Narrowing of a vessel produces a similar effect, by increasing the friction of the blood against the walls at the constricted point.* Murmur of both these kinds is always synchronous with the pulse, and not rough, unless the blood be spanæmic, and the inner walls deficient in smoothness.

499. (2) Mere atheroma, unless accumulated in unusually large quantity, does not produce murmur; infiltration with induration-matter, by puckering the inner surface irregularly,

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* Dr. Corrigan, however, argues with great ingenuity that it is not at the constricted point of a vessel, but within the area of the loose walls beyond, that murmur is produced.

roughens the sound; erosions of the lining membrane do so more effectually; and calcification of the vessel, if it cause irregular elevations of that membrane, is still more efficient. Murmur is also produced by particles of exudation-matter studding the surface of an artery recently inflamed. This variety of murmur is always synchronous with the pulse.

500. (3) In the varieties of aneurism with diseased coats, form and surface are changed so as to produce murmur, which may be systolic, diastolic, or both, and attended or not with sound also. The conditions regulating the time and accompaniments of this murmur, will be described under the head of Aneurism.

501. (4) Intra-thoracic varicose aneurism, in all its forms, is attended with murmur synchronous with the arterial diastole, sometimes prolonged through its systole.

502. A certain amount of force in the heart's action is essential to the generation of arterial murmur; increase of that force will convert a soft into a harsh quality instantaneously.

503. Murmurs heard in the thoracic aorta, single or double, are often merely conducted from the heart. But if a murmur, audible at any part of the arch, be of different pitch, of greater intensity, and of harsher quality than a synchronous murmur at the aortic base, it may be inferred that a cause of reinforcement exists in the vessel itself. The only source of fallacy would be the chance coexistence of badly conducting materials over the base of the heart, and of excessively good ones over the arch of the aorta. The characters of an arterial murmur will sometimes guide the observer partially to a knowledge of its anatomical cause; but the actual determination of this will mainly turn on the state of other physical signs.

504. *Inorganic* arterial murmur is commonly softly blowing, if the vessel be auscultcd without pressure. If pressure be used, it rises in pitch, and becomes sharply whiffing or whipping, resembling the sound produced by a quick stroke of a riding-whip through the air. It is intermittent, never double, never continuous, never synchronous with the systole of the vessel,

and affects the arterial system extensively, instead of being purely local, as the organic variety.

The clinical conditions of inorganic arterial murmur, are certain of those of cardiac murmur of the blood-class, especially spanæmia: I regard it therefore as a hæmic murmur. It is said that plethora produces it;—a statement I have been unable to verify clinically. But of this more by and bye.

505. Midway between the organic and inorganic varieties, stands the murmur of an artery, healthy in itself, but pressed upon by an adjacent tumor. Arterial murmur of this mechanism may synchronise with the systole of the vessel.[*]

VENOUS SYSTEM.

SECTION I.—INSPECTION.

506. Considerable distension of any particular portion of the venous system indicates the existence of obstruction in the connected main trunk, or in the right side of the heart itself.[†]

The internal and external jugular veins are the veins most frequently found enlarged,—the right more commonly than the left, when one side only is affected. This obstructive distension, uniform or varicose, even if increasing the size of the external jugular almost to that of the little finger, is unattended either with change in the integuments, hardness or cordiness of the vein, or tenderness under pressure. The common causes of this condition are tricuspid regurgitation, and pressure on the superior cava or innominate veins by intra-thoracic tumour or aneurism; more rarely, simple dilatation of the right cavities of the heart.

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[*] Gosling, U. C. H., Females, vol. v., p. 130.

[†] It is not intended here to refer to the signs of local diseases of the veins, such as phlebitis or varix.

Excess of enlargement on one side cannot be implicitly trusted to as significant of local pressure, instead of cardiac disease, as the cause of enlargement. Dilatation of the heart and tricuspid regurgitation may be attended with excess of enlargement of the left, as well as of the right, external jugular.

507. The external jugular vein, oftener the right than the left, is occasionally the seat of visible pulsation, especially at its lower part near the clavicle. Irregular in amount and in rhythm, though obviously connected in the main with the ventricular systole, jugular pulsation wants the distinctness of an arterial pulse, and is rather an unsteady intermittent tremulousness than a series of well-defined beats. The effect of inspiration and expiration on the blood in these veins partly explains the irregular rhythm; which may also, in part, be referred with extreme probability to the influence of the auricular systole. The impulse producing it comes visibly from below; and when a portion of the vein is emptied by pressure from the clavicle in an upward direction, it re-fills immediately from below, while the pressure is sustained above.

Lancisi, the original observer of this phenomenon, supposed that it was produced by eccentric hypertrophy of the right ventricle. Hope, holding to this view, explains the impulse by the "impetuous recoil of the tricuspid valve," which repels the blood about to descend into the ventricle with such force that its impulse is propagated back to the jugular veins. Many persons maintain that jugular pulsation only occurs where the tricuspid orifice is too much dilated to admit of closure by its valve, whence ensues regurgitation into the veins during the ventricular systole. Dr. Parkes teaches that, in addition to tricuspid insufficiency, rupture of the valves at the junction of the internal jugular and subclavian veins is a necessary condition of the phenomenon.

I know of no facts positively showing the necessity of such rupture of valves; the vessels may be sufficiently distended to render their valves incompetent, which is all that is required.* The valves, too, may be congenitally absent. Further observation on these points is, however, desirable. But, as concerns the tricuspid orifice? Unquestionably jugular pulsation is most frequently met with in cases of tricuspid insufficiency, though by no means in all of the class; while, as I have decidedly observed it, where the valve was not demonstrably incompetent, in cases of dilated and hypertrophous right ventricle, I cannot refuse to admit that this latter condition alone may produce it.† If the ventricle be hypertrophous, and the valve insufficient, the pulsation reaches its maximum. It is to be remembered, too, that respiration affects jugular pulsation, emptying the vein in inspiration, distending it in expiration. The parts played severally by respiration and cardiac action, may be distinguished by causing the patient to suspend his breath for a moment.‡

Extreme jugular pulsation may exist without the least visible throbbing of any other vein.

508. The right mammary veins may be knotty and pulsatile; I have seen this in cases of insufficiency of the tricuspid valve, and of intra-thoracic tumour.

509. But veins, much more distant from the heart than these,—the veins, for instance, of the *dorsa of the hands and feet,*—may be the seat of pulsations either of cardiac or of

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[*] Thos. Denham, U. C. H. Feb. 1851, Males, vol. vi., pp. 69, 77. Here notable pulsation, both of the jugular and innominate veins, had existed during life; the valve was ascertained to be perfect, but enlargement of the calibre of the veins had obviously rendered it incompetent.

[†] It is too constantly assumed by observers, that where the tricuspid valve is insufficient to close the orifice at death, it has been so during life also. What proof have we that an instinctive constriction of the orifice does not accommodate the width of the opening to the capabilities of the valve?

[‡] The jugular pulsation sometimes noticed in pneumonia may depend, either on cardiac influence, or on that of respiration and the pulsation of the contiguous artery combined; the fluttering movement of the vein on the affected side sometimes attending pleuritic effusion is, as far as I have seen, of the latter mechanism,—and may totally disappear in the erect posture, though well marked in decumbency (I. Harrison, U. C. H., Females, vol. ix., pp. 329, 345).

respiratory rhythm, or of both combined. Dr. Jenner has very kindly favoured me with the particulars of three cases illustrating these various rhythms. When the rhythm is cardiac, pressure on one of the pulsatory veins on the back of the hand, increases the strength and distinctness of the pulsations (which equal in number those of the radial pulse) to the distal side of the point pressed on, annuls them to its proximal side; the respiratory movements exercise no influence on the pulsations. When the rhythm is respiratory, the vein collapses in inspiration rapidly, swells in expiration slowly; and, when pressed on, its pulsation ceases to the distal, increases to the proximal, side of the point pressed on. One of Dr. Jenner's cases exhibits the co-existence of the two sorts of pulsation (respiratory,—and cardiac, by *vis à tergo* through the capillaries, probably) in an infant aged eighteen months, cut off with pneumonia secondary to tubercles. The reason why respiration and cardiac action should exercise this influence on distant veins in some cases of disturbed thoracic action, and not in others, apparently similar, is yet to be discovered.

510. In all cases of apparent venous pulsation, the possible influence of accompanying arteries must be inquired into.

SECTION II.—APPLICATION OF THE HAND.

511. Thickening of the walls of the jugular veins sometimes arises in cases of tricuspid regurgitation of long standing. If in such a case those vessels pulsate, it is very probable their diastole will be perceptible to the fingers; but I do not remember ever to have actually observed this.

512. Visible pulsation is occasionally attended with soft thrill,—a minor degree of the arterial phenomenon of the same name.

SECTION III.—AUSCULTATION.

513. The venous system, as was originally and most ingeniously shown by Dr. Ogier Ward, is the occasional seat of

audible murmurs, which possess one invariable character—that of *continuousness*.

514. In point of quality, venous murmurs are referrible to four types: the blowing, the whistling, the humming, and the modulated. The blowing varieties may be as soft as the respiration-sounds in health, sharply blowing, loudly blowing, as the sound heard on applying a shell to the ear, or actually roaring. Or, the murmur may be cooing or whistling. To the humming type belong various murmurs resembling more or less closely the noise of a humming-top, the buzzing of a fly, the singing of a tea-kettle, &c. Lastly, venous murmurs are sometimes distinctly modulated, consisting of a series of separate tones, capable of musical notation, recurring at tolerably regular intervals, and accompanied by a low hum, which gives the continuous character to the whole.

Inclining rather to softness than roughness, and of moderate intensity, inaudible unless the ear or stethoscope be applied directly to the surface, generally of low pitch, as the whispered word *who*, (when modulated, of course, this is variable,) venous murmur is liable to change in intensity and quality from one moment to another. This change occasionally occurs from some intrinsic untraceable agency; more frequently from some one of the following causes. Acceleration of the circulation intensifies venous murmur; and as inspiration favours the rapid flow of blood in the veins adjoining the thorax, in these veins, at least, that act ought to, and does actually, increase the loudness of an existing murmur. But, on the other hand, suspension of the breath at first exercises even more markedly the same effect; the sharp collision of the blood disks *inter se*, and against the walls in the struggle to move onwards, and the increased vibration of the walls themselves, probably explains this. If the breath be held for any time, the murmur disappears. Any posture which stretches moderately the vein under examination, intensifies its murmur: if the part examined be a muscular one,—the thigh, for example,—there is a source of fallacy in the rumbling sound of muscular contraction, which must be guarded against by auscultation in a perfectly relaxed condition of the muscles. In the neck, murmur is stronger in the erect than in the lying posture; probably from the greater rapidity of flow in the former attitude. Venous murmur attains its maximum under a certain amount of pressure, ascertainable in each instance only by actual experiment. Less or more pressure weakens and finally obliterates all audible sound. Sex exercises no influence on the intensity of venous murmur, nor, directly, on its frequency. No doubt, it is greatly more commonly observed clinically in females than in males; but this depends simply on the disproportionate frequency of its physical conditions in the two sexes. No evidence has ever been adduced, showing that a given state, which fails to generate murmur in a male, will succeed in the case of a female.

515. The veins in which murmur occurs, may, as far as I have observed, be arranged, as follows, in order of frequency. The external and internal jugulars, on both sides, or on one side only, in the latter case most frequently the right; the subclavian veins; the femoral (I have never failed to find it in these vessels when well developed in the neck, and it may be caught in them sometimes when inaudible in the jugulars); the axillary; the superior cava and innominate veins;* the veins of the bend of the elbow; certain abdominal veins;† the pulmonary veins; and the superior longitudinal sinus, especially at its termination in the torcular Herophili.‡

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* M. Clarke, U. C. H., Females, vol. iv., p. 261, Oct. 1848, "Lancet," March 31, 1849.

† Sus. Roberts, U. C. H., Females, vol. v., pp. 218—220, Oct. 1850. Continuous loud "roaring" hum, coupled with arterial intermittent murmur, of maximum strength a little above and to the *left* of the umbilicus, but audible even to the right of the middle line. Here, too, appear cases of continuous hum, audible on deep pressure at the right edge of *some* enlarged spleens.

‡ Davis, U. C. H., Females, vol. iv., p. 138. In this case a continuous remittent murmur was also audible at both sides of the mid-dorsal spine.

516. Invariably continuous and never intermittent in rhythm, murmur in a vein may be simply continuous, that is, of equable force constantly; or it may be remittently continuous, undergoing reinforcement and weakening at regular intervals. The type is humming when the rhythm is thus remittent. Two causes of this remittent character have been suggested: the pulsations of an adjoining artery against the soniferous vessel, which give at regular intervals a momentary impetus to the current in the interior of the vein; and the co-existence of ordinary intermittent blowing murmur in the accompanying artery. The majority of instances are fairly explicable on one or other of these principles; some, which are not so, may, it would appear, be explained by intrinsic inequality of force of current in the veins,—an inequality, which, we know, positively exists in cases of venous pulse.

517. Venous murmur may be accompanied or not with arterial or cardiac murmur, the latter basic in seat and systolic in time.

518. But do the murmurs, just described under the name of venous, really deserve the title? The history of opinion on the point is exceedingly curious. Long after the venous site of the "humming-top" murmur in the neck had been demonstrated to the satisfaction of observers in this country, M. Bouillaud, and French writers generally, persisted in localising it in the carotid arteries. Skoda treating of it so lately as 1842, in his section on arterial murmurs, likewise placed it in the carotid, and actually ridiculed the idea of venous origin.* And now that he has adopted the correct view, his countryman, Kiwisch, experimentalises, and writes, to prove that the veins are never the seat of murmur, the carotids being the real site of the cervical hum in a chlorotic woman. Kiwisch's argument, however, seems to me simply to present one more illustration of the unfortunate facility with which, in matters

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* Percussion, &c., 2te. Aufl. p. 199.

medical, ingenuity may succeed in a plausible effort to make "the worse appear the better reason." The following, among other facts, appear, in truth, conclusive as to the venous origin of these murmurs.

1. The gentlest pressure on the external jugular above the point on which the stethoscope rests, instantaneously silences the hum beneath,—an amount of pressure so slight as to be incapable of exercising the least influence on the neighbouring artery. 2. Continuous hum and either arterial sound or arterial intermittent blowing murmur may in some cases be heard at one and the same moment separately; I have repeatedly noticed this in the femoral artery and vein. 3. Continuous hum can be heard, where there is no artery to furnish murmur,—as, for instance, along the longitudinal sinus and at the torcular Herophili. 4. The erect posture increases, the lying posture lessens continuous murmur in the neck; the precise converse effects are observed below the groin: this is only intelligible by the changes in the venous current produced by altered postures,—altered postures which exercise no appreciable influence on arterial action. 5. Continuous murmurs of different quality and different pitch can occasionally be heard over the external and internal jugulars; there is no difference of artery in the two situations.*

519. Various hypotheses suggest themselves in explanation of these murmurs, referring them severally to altered composition of the blood, diminution of the mass of the blood, change in the blood's motion, and change in the dynamic and physical condition of the walls of the veins.

(*a*) Venous murmurs are so constantly connected clinically with chlorosis and other maladies, of which poverty of blood forms an element, that their generation in some way or other though that condition of blood was generally, and without, it must be confessed, actual proof, accepted as a fact. The deficiency of red disks in anæmia, hydræmia, and spanæmia was held to explain the noisy movement of the blood,—a notion which received its first contingent of precise support from M. Andral, who, attempting to establish the exact relationship between the amount of spanæmic change and the constancy of venous murmur, finds that if the red corpuscles fall below 80 per 1000, murmur is constant; if they range between 80 and 100, pretty frequent; if between 100 and 115, occasional; if between 115 and 126, very rare; if they reach the average of health, invariably absent. It is argued that the blood being unnaturally thin, the friction attending its movement is, according to a law of Poisseuille's, increased to such an amount as to engender sound.

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* Perhaps it is not altogether unworthy of notice, also, that Kiwisch failed to produce continuous murmur in the arteries laid bare, and auscultod under various amounts of pressure in large-sized animals.

But there are some facts apparently subversive of this theory. Thus it is well known that in cases of chlorosis treated with iron, colour returns to the tissues, long before venous murmur disappears; and, *vice versâ*, Becquerel and Rodier give analyses of the blood of two chlorotic girls, presenting well-marked venous hum, with a mean proportion of 125·1 per 1000 of red corpuscles,—certainly an amount falling within the limits of health. It was long since stated, too, by the "London Heart Committee," that murmur may be produced in the veins by pressure in a state of robust health; and I have heard it in women of florid complexion, who, as far as I could ascertain, had never been *symptomatically* anæmic.*

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* Winterich, "Brit. and For. Med. Chir. Rev.," April, 1852, found the per centage of venous murmurs in *healthy* persons of both sexes as follows :—

Age.	Males.	Females.	Age.	Males.	Females.
1 — 5	97	98	30 — 40	80	86
5 — 10	94	95	40 — 50	77	78
10 — 15	89	95	50 — 60	72	75
15 — 20	86	88	60 — 70	68	71
20 — 25	82	88	70 — 80	40	39
25 — 30	80	86			

Dr. H. Davies' inquiries confirm to a certain extent these results.

Is it possible that increase in the proportion of white corpuscles, a very positive attendant on chlorosis, and, as shown by Remak, on repeated hæmorrhage, plays a part in generating venous murmur,—such increase probably entailing increase of friction and labour in the circulation? It is certain that with disappearing anæmia and lessening hum the white corpuscles may be found to diminish; * but of course association does not prove causation.

(*b*) Pure diminution in the mass of the blood explains jugular hum according to Hamernjk, by the whirling and eddying motion into which the dwindled venous current is thrown in passing along a vessel, which in consequence of its anatomical arrangement always retains a certain width at its lower part. But it seems impossible to prove this alleged influence of anæmia to the exclusion of spanæmia, inasmuch as diminution of the blood's mass seems never to take place without a fall in the proportion of red corpuscles.

(*c*) The influence of eddying and interrupted motion, however, seems difficult to contest in face of the manifest part played by regulated pressure with the stethoscope in eliciting the murmur.

(*d*) If the veins contain less blood than natural, their walls become looser,—the increased facility of vibration, thus arising, accounts for the murmur in the opinion of some. M. Vernois, in a modified form of this theory, looks on the prominent folds, into which the walls of the veins are thrown, as the cause of murmur, through the impediments they offer to the flowing blood. But as the sinuses of the dura-mater, with their quasi-rigid walls, give murmur, the non-necessity of such obstructive action is obvious.

520. The real mechanism of these murmurs is consequently obscure enough. It seems to me impossible to ignore the influence of the composition of the blood; in highly marked

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* M Pitt, U. C. H., Females, vol. viii. pp. 21, 57, Nov. 1851.

spanæmia, neither flaccidity of veins, pressure on their surface, nor even velocity of current, is required for the generation of murmur,—it exists in the sinuses of the dura-mater, especially where the arrangement of these canals is such as to promote abrupt collision of opposing currents meeting at a conflux, as at the torcular Herophili.

521. It is held by some writers, that as murmurs may be heard in the veins of healthy looking persons, they cannot be accepted as evidence of a morbid state of the blood. Such "normal" venous murmur is, as far as I have observed, always very feeble in the adult. And, inasmuch as venous murmur cannot be found by any means in all adults, there is clearly something unnatural in those who present it. Even in children it cannot invariably be caught; and what is healthy in a child may be morbid in an adult,—take the single instance of the different states of Peyer's glands in childhood, and more advanced life. The composition of the blood differs in infancy and adult age; it acquires new characters, again, in old age, and these characters may very possibly render the fluid less apt to generate sound, whence the less frequency of venous murmur in old age.

I would sum up on this matter, clinically, by saying, that a state of blood in which the venous current becomes slightly soniferous, is not incompatible with apparent health; but that intense, readily audible and diffused venous murmur, is characteristic of those morbid conditions of the fluid, called hydræmia, anæmia, and spanæmia.

523. The diagnosis of venous murmurs turns essentially on their continuous character; and is excessively easy, except when accidental circumstances occur to render that character obscure. This happens sometimes about the base of the heart anteriorly, and between the scapulæ in the back. Pulmonary venous murmurs are partially masked by the cardiac sounds.* In addition to its peculiar quality, pitch, inconstancy, ready

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* Commentary on case of Clarke, "Lancet," March, 1849, p. 332.

influence by change of posture, as guides to the venous origin of the murmur, its rhythm in respect of the heart's beat will *sometimes* aid in connecting it with the veins. Thus a diastolic murmur at the base (the signs of *organic* disease at the cardiac orifices being deficient) must be venous, according to my experience;—at least an inorganic cardiac murmur of that site and rhythm has never fallen under my notice.

524. If a lateral communication exist between a contiguous artery and vein, so as to permit the current from the former to enter the latter, a murmur of whizzing or whirring character, and essentially intermittent, results.

PART II.

DISEASES OF THE LUNGS, HEART, AND GREAT VESSELS.

CHAPTER I.

THE LUNGS AND APPENDAGES.

SECTION I.—THORACIC PARIETES.

I. PLEURODYNIA.

525. By pleurodynia, is understood rheumatism of the walls of the chest, affecting especially their muscular and fibrous textures. Most common on the left side, and in the infra-axillary and infra-mammary regions, its essential feature is pain, more or less acute, sometimes of agonising severity, increased by deep inspiration, by coughing, by movements of the trunk, and even of the arm,—by decumbency on the affected side, and by pressure both on the ribs and in the intercostal spaces.

526. Unless it coexist with acute articular rheumatism (and, as far as I have seen, the combination is excessively unusual), pleurodynia is unaccompanied with fever.

527. The play of the chest on the affected side is interfered with. The movements of expansion and of elevation are diminished in freedom, and their rhythm becomes jerking: in

the female this is somewhat less perceptible than in the male, when the affection occupies its ordinary seat,—the inferior regions of the chest. The natural relationship of the abdominal and costal movements in forced breathing becomes perverted. [80]. The respiration-sounds are of intermittent weak type, and jerking rhythm; there is no friction-sound to be heard. If the patient can be induced to expand the side fully, the percussion-sound proves natural; even when the side is most inactive, it is difficult clinically to discover the diminished clearness of note, theoretically to be expected.

528. Pleurodynia simulates the congestive, or dry, stage of pleurisy. Friction-sound is not, on the first onset of pain, of as much use as might be anticipated in the distinction of the two affections;—for, on the one hand, friction-sound may be wanting on the invasion of pleuritic inflammation;—and, on the other, the jerking rhythm of pleurodynic respiration may so closely imitate the grazing variety of that sound, as to leave a cautious observer in doubt. If with pleurodynia there be a chance co-existence of febrile action, cough and slight bronchitis, a positive diagnosis should be refrained from, until, a certain number of hours having elapsed, the friction-sound of prominent pleural capillaries or of exudation-matter, if the case be one of pleurisy, will have established the fact beyond the possibility of doubt.*

The pain of pleurodynia is sometimes seated so low down, as to lead to the suspicion of peritonitis. The absence of rigors and severe febrile symptoms generally, as also of vomiting, of anxious facies and other signs of deep constitutional shock, will distinguish the former from the latter disease. In both, the abdominal walls are tender to the touch; but the pain of pleurodynia is more fixed, and relatively more increased by voluntary movements, than that of peritonitis.

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* In a case seen with Dr. Neil Arnott, the diagnosis was rendered difficult by the impossibility of making a physical examination of the chest, so exquisite was the tenderness. But, on the other hand, the very intensity of the pain and tenderness argued in favour of pleurodynia rather than pleurisy; and the event proved that the pleura had escaped. In this instance there was articular rheumatism also.

529. True rheumatic pleurodynia yields rapidly to cupping, dry cupping, anodyne and stimulant liniments, and the use of colchicum and an alkali.

II.—INTERCOSTAL NEURALGIA AND NEURITIS.

NEURALGIA.

530. The intercostal nerves—especially on the left side, and from the sixth to the ninth—are not unfrequently the seat of neuralgia, occasionally of actual inflammation.

531. The pain of this neuralgia is severe, varies in precise character, as that of similar affections elsewhere, occurs paroxysmally, follows the course of the affected nerve, or seems to pass directly backwards from the edge of the sternum to the vertebral groove, and is frequently accompanied with pain in the branches of the brachio-cephalic plexus. Generally speaking, three tender points (as was first, I believe, shown by M. Valleix) may be detected by pressure in the course of the affected nerve or nerves,—one in the vertebral groove, another about the axillary region, a third in front towards the terminal ramusculi.

582. The physical signs are the same as those of pleurodynia; impaired thoracic movement, with weak jerking respiration,—the percussion-signs being negative.

533. Intercostal neuralgia is, in the female, often associated with that of the mammary gland,* and spinal irritation; and it often occurs in an obstinate form as a sequence of herpes zoster. But the complaint derives its interest, in respect of pulmonary pathology, from the possibility of its being taken for an evidence of pleurisy, and from its being pretty frequently associated with

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* Benyon, U. C. H., Females, vol. viii., p. 225, Martin, U. C. H., Females, vol. ix., p. 190.

phthisis. Its relationship to pseudo-anginal affections of the heart will be elsewhere considered.

534. The three painful points in the course of the nerve indicate the true nature of the disease, distinguishing it from pleurodynia, costal periosteitis, and all pains of intra-thoracic origin.

535. If the tenderness be extreme at any one of the three points referred to, a few leeches are requisite; subsequently flying blisters, will, as concerns local measures, often complete the cure. But sometimes, after a sharp attack, a minor amount of pain recurs from time to time, which is best relieved by the endermic use of morphia, or by inunction with ointments containing belladonna or aconite, or in severer cases their alkaloids. The internal treatment will vary according to circumstances. If the neuralgia appear a mere subsidiary phenomenon of phthisis, there is no necessity for altering the treatment designed for the main affection; if, on the contrary, it become for the time the prominent disease, quinine, arsenic, and iron, combined with purgatives, are, as in the case of other neuralgiæ, advisable.

NEURITIS.

536. Inflammation of the intercostal nerves, with vascular injection, and considerable enlargement of their substance, has been described by M. Beau, as an attendant on pleurisy both in its acute idiopathic, and chronic tuberculous forms. The pleuritic "stitch" in the side he looks upon as the result of such neuritis. Further observations on this subject are called for: that "stitch" may have existed without anatomical evidences of neuritis being detected after death, is indubitable.

III.—VARIOUS PARIETAL DISEASES.

537. A number of affections of the walls of the chest produce physical signs that might, without due caution, be confounded with those of intra-thoracic diseases. A mere

reference to some of them, — such as may serve to put the physician on his guard—is all that can be ventured on here.

538. Thus costal periosteitis, with its tenderness, local percussion-dulness, and somewhat weakened subjacent respiration, might very readily be mistaken, at the apex of the chest, for consolidating pulmonary disease beneath, unless the ribs were carefully examined. Carcinomatous infiltration of a rib, especially if there be no external prominence, may in like manner deceive.* Abscess of the sternum,† or situated between the periosteum and the anterior,‡ or at the posterior, § surface of that bone, may simulate, according to its precise site and characters, mediastinal abscess or tumour, or aortic aneurism. Subcutaneous emphysema and anasarca sometimes give rise, through the movements of respiration, to rhonchoid noises simulative of true bubbling rhonchus and friction-sound [232].

SECTION II.—BRONCHIAL TUBES.

INFLAMMATION.

539. Inflammation of the bronchial tubes, or bronchitis, the most common of pulmonary diseases, occurs, idiopathically, in the acute and chronic forms.

I.—ACUTE BRONCHITIS.

540. Clinically, as well as pathologically, acute bronchitis differs so materially, according as the disease implicates merely the larger and medium-sized tubes, or involves the capillary ramifications and confines of the air-cells, that the two varieties must be separately considered.

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* I have seen two contiguous ribs united in this manner.

† Reilly, U. C. H., Males, vol. vii., p. 305.

‡ Mottlee, U. C. H., Females, vol. v., p. 36.

§ Buckley, U. C. H., Males, vol. ix. p. 248, 310.

A.—Bronchitis of the Larger and Medium-sized Tubes.

541. This variety of the disease is anatomically characterised by injection of the mucous membrane, capilliform generally, maculated occasionally, and rarely visible to the naked eye beyond the third or fourth divisions. Commonly strips of good length are obtainable; but in rare instances they are short from slight softening: the membrane is in exceptional cases thickened, and the sub-mucous tissue infiltrated with soft exudation-matter. Epithelial desquamation occurs in patches: true ulceration never. The form of the tubes is unaltered: they contain aërated mucus, exudation-matter non-moulded, pus and epithelium,—the whole free from blood. The pulmonary tissue is natural.

542. The invasion of the disease is commonly marked by coryza, sore throat, and slight hoarseness, chilliness, scarcely amounting to rigors, with lassitude, aching pains in the limbs, and frequent pulse. The occurrence of coryza is significant of the primary character of the disease,—tuberculous bronchitis very rarely originates with this symptom.

543. The disease being established, more or less discomfort and pain are felt behind the sternum,—a sensation of heat, soreness, or rawness of the bronchial surfaces, increased, perhaps, to acute pain by coughing, and attended with a sensation of oppressed breathing. The respiration is increased in frequency, slightly out of proportion with the pulse,—in severe cases, notably so. The cough, an essential feature of the disease, at first short and dry, or nearly so, occasionally paroxysmal, and severest after sleep, loud, hoarse, and ringing, is attended, after the lapse of one or two days, when it becomes loose, with expectoration of frothy mucus, watery in the main, ropy in some measure, of saline taste, faintly yellowish, yellowish green, or grayish yellow colour, free from blood visible to the naked eye, varying greatly in quantity, and gradually becoming muco-

purulent. The sputa run together into a single mass, except in rare instances, when they present the nummulated form, with perfect opacity: I have seen this, even in children, in the bronchitis of measles. Referred sometimes, by the patient, to the sternal region, the cough is more frequently brought on by a tickling feel about the trachea and larynx, where the patient will consequently obstinately contend his whole ailment lies.

Microscopically, the clear sputa of bronchitis consist of hyaline fluid, mucus-corpuscles and small-sized tesselated, cylindrical and ciliated epithelium; the opaque variety, of exudation-cells and masses, epithelium, pus-cells, and some rare blood-discs. Saliva and epithelium from the mouth are accidental admixtures.

544. Lasting from four or five days to two or three weeks, in cases of complete recovery (which make the vast majority), attended with febrile action of sthenic or asthenic type, the acute disease may besides terminate in its chronic form, or actually prove fatal. The variable length of the attack in the first class of cases depends sometimes on the extent of surface involved; probably sometimes on the depth to which the disease reaches in the bronchial walls; but also in many cases on the existing diathetic state, inasmuch as with the same general and physical signs, and under the same treatment, cases vary pretty widely in their duration. The chronic disease supervenes more especially, where the deep tissues of the tubes are involved by the acute attack. Death, solely occurring in infants, in aged persons, and in those constitutionally debilitated by excesses, injuries or disease, is even in these classes excessively rare; acute bronchitis, really destructive of life, belongs to the next variety.

B.—Capillary Bronchitis.

545. Even in this variety of the disease the proper tissue of the minor and capillary tubes suffers less than that of the larger. Still, redness, irregular thickening, and in some cases

softening, in yet rarer ulcerations, may be detected with the naked eye in the smallest bronchi thus traceable. The tubes are very generally dilated uniformly, or more rarely unevenly; they contain even to their very extremities muco-pus and exudation-matter more or less closely adherent,—the latter in the form of casts, never of any great length, and either solid or tubular.

The lungs are generally air-distended and sometimes acutely emphysematous: hence general excess of bulk. But here and there the surface is depressed in the site of collapsed lobules, airless and quasi-solid, yet capable of insufflation from the trachea, unless the exudation-matter in the communicating tubes be very abundant. True pneumonia, lobular or diffused, is of purely exceptional occurrence;* the parenchyma is often even unusually pale. Minute accumulations of pus, looking intra-parenchymatous, but really traceable to the interior of the air-cells and ultimate tubes, are sometimes found towards the surface of the lung.† The bronchial glands are often inflamed,—injected, large and soft.

546. Capillary bronchitis being generally preceded by inflammation of the larger tubes, the symptoms of invasion are not so decided and severe as might be expected. Rigors are rare; vomiting rarer. Still the capillary tubes may suddenly become involved, even after a lull of symptoms of the milder disease; and though even under these circumstances the rigors of an acute seizure may be wanting, the severity of the general symptoms from the first shows the gravity of the attack. On the other hand I have known well-marked rigors, vomiting, and headache occur at the outset.

547. Essentially asphyxiating in its effects, as the anatomy

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* This is especially true in the adult; but, even in the infant, the frequency of lobular pneumonia has been greatly exaggerated by some writers, in consequence of their mistaking collapsed for inflamed lobules.

† These are the "purulent granulations" of Rilliet and Barthez; for the form of the disease attended therewith, they propose the rather contradictory title, "vesicular bronchitis."

of the disease prepares us to expect, capillary bronchitis is attended with variable congestion of the external surface, lividity of the lips, cheeks, tip of the nose, external ears, finger-ends, and with fulness of the jugular veins, indicating obstructed circulation through the right side of the heart, sequential to that of the lungs. The pulse and respiration are both extremely frequent,—the latter out of proportion to the former, but, as far as I have seen, never to the extent observed in some cases of pneumonia. Dyspnœa, it may be carried to orthopnœa, and paroxysmal; sense of oppression and stuffing in the chest; cough at first almost perpetual, with exacerbations from time to time, in exceptional cases infrequent; post-sternal pain, sharp, aching, sore, slight or null; expectoration sometimes muco-purulent, yellowish green, or bright green, opaque, very abundant and free, at other times viscid, ropy, glutinous, containing exudation-matter in patches* —the whole attended with feeble reaction, hot, warm or about natural temperature of the skin, which is sometimes moist, but free from sudamina, anxious countenance, and extreme general restlessness,—constitute the ordinary symptoms.

In cases tending to a fatal issue, exhaustion soon ensues. The pulse, if at first of some power, rapidly loses its strength, becoming excessively frequent,—120 to 150, but free from irregularity; the respiration, varying commonly from 36 to 50, maintains its relative frequency, almost until the closing struggle.

Pulse-respiration ratios of 3.0, 2.5 or 2.25 to 1 are not uncommon; but sometimes, even so much as forty-eight hours before decease, the frequency of the respiration falls while that of the pulse continues to rise: under these circumstances I have known a ratio of 4.5 to 1, that of health, coincide with a pulse beating 144 per minute.

As long as his strength permits, the patient sits erect or bent forwards; but the body gradually yields; and it is not uncommon to find patients, while still perfectly conscious, lying with the head lower than the shoulders. The sputa gradually diminish in quantity, from failure of power to expectorate; the skin, generally livid or cyanotic in tint, falls in temperature, and becomes covered with cold clammy perspiration, sometimes rather copious, rarely attended with sudamina; the breath grows cool; fitful dozes lapse into a state of somnolence; muttering delirium, in some instances slight convulsions, precede a comatose state which is the immediate forerunner of death. The fatal termination is sometimes hastened by the accidental blocking-up of a large bronchus with secretion.

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* I have seen white patches of the kind in the sputa of a patient aged sixty; in infancy it is not very uncommon.

548. The prognosis of capillary bronchitis is very serious. Robust adults may, it is true, generally be saved by active measures; but of young children and aged persons (even though free from chronic bronchitis) attacked, it has been calculated from three-fourths to one-half perish. Death generally occurs between the eighth and the twelfth days; convalescence sets in, in cases of recovery, between the tenth and twentieth. I have known life destroyed in the adult in forty-six hours. If the alimentary canal chance to be affected at the same time—if there be gastric or intestinal irritation, the vital powers fail with even unusual rapidity.

The indications of fatal tendency in individual cases are suppression of expectoration, without improvement in other symptoms, increasing viscidity of the sputa, excessive frequency of pulse and respiration, failure of heat, clammy sweats, and somnolence.

That slackened and comparatively quiet respiration does not in itself justify a favourable prognosis, where other symptoms remain unrelieved, is inferrible from what has already been said.

549. The physical signs of both the varieties of simple acute bronchitis may be considered together.

Little of a practically valuable kind is discovered by inspection in slight bronchitis: the form of the chest is not visibly altered; and, unless there be emphysema present, the perversion of movements is insufficient to attract the eye. If the disease be severe, the costo-abdominal expansion-movements assume the characters of healthy *forced* breathing; the costal increase in amount, the abdominal decrease. Where the dyspnœa is extreme, the lower end of the sternum and connected cartilages sink in with inspiration. The expiration-movements are slow, laboured, and inefficient. The hand, applied to the surface, occasionally detects distinct rhonchal fremitus,—its presence furnishes a rough guide to the seat of the rhonchus, as it will scarcely be transmitted to the surface, if a deep bronchus be the source of vibration. It may be more distinctly perceptible during inspiration than expiration, or *vice versâ*. Dr. Stokes believes that it is more marked in the child and female than in the adult male, and at the middle and inferior parts of the chest than the superior. I have found it very remarkably developed in infants of from six to twelve months old. The state of vocal fremitus varies; it sometimes exceeds the average of health.

The percussion-sound, as a rule, is not, practically speaking, impaired in clearness, and may be even a little raised above the pitch of health. I have known this excess of clearness maintained in the central regions of the back, in capillary bronchitis, through the whole course of the disease, and even within a few hours of death. Besides, the area of clear pulmonary resonance extends in front a little further downwards than natural—a fact easily ascertained over the liver—and expiration has less effect than in health in diminishing the superficial extent and amount of that resonance. The lung is held to a certain extent in a state of mechanical distension, from diminished elasticity of its tubes and substance, and from imprisonment of air by glutinous mucus.

In certain cases, slight dulness under percussion may be detected, especially at the postero-inferior parts of the chest. But this condition of sound in simple bronchitis is singularly rare; and when we reflect that considerable turgescence and thickening af the mucous membrane over a large extent of surface form part of the anatomical features of the disease, the usual clearness of resonance affords fair ground for surprise. The fact of such clearness existing is an important one; as it will commonly enable us to infer the idiopathic character of the disease, and conclude without hesitation that it does not depend upon or attend tuberculous deposition.

The imperfect resonance in exceptional cases is variously produced. Sometimes depending upon accumulation of bronchial secretion, it is then chiefly observed at the base and posteriorly, and occurs more especially in subjects of debilitated constitution, or in those labouring under prostrating diseases,—as, for example, typhoid fever—of which the bronchitis is only a secondary condition. In these cases there is often some congestion, or more rarely œdema, of the lung, which takes its part in producing the deficiency of clearness. Occasionally the dulness seems traceable to collapse of the lung, consequent on pressure on a main bronchus by enlarged bronchial glands,—the bronchitic secretion contributing its share, at the same time, of defective resonance.*

Reference has already been made [137] to the occasional occurrence of a pseudo cracked-metal resonance in the bronchitis of young children especially. I would add, to what is there said on the subject, that unlike the true cavernous sign, this simulation of it is changeable in place.

By auscultation, we learn that true respiratory sound, weakened, sometimes, even to suppression, in the tissue communicating with the affected tubes, is exaggerated on its confines and elsewhere,—hence, especially so in the upper parts of the chest. The respiration is coarse and noisy, often more like dry rhonchal, than true breathing, sound; whence I have been in the habit of calling it "sonorous respiration." The sounds, dry and harsh in quality, are accompanied, and it may be masked more or less perfectly, by sonorous, sibilant, and bubbling rhonchi, in various combinations,—the former often of musical quality. Expiration is sometimes much prolonged, and, as it were, laboured. The vocal resonance is not perceptibly affected as a rule; but in some cases, probably from nasal character in the voice, it has a sniffling quality.

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* Ransom, U. C. H., Females, Nov. 1848.

As a general fact, the sonorous and sibilant rhonchi are most marked and constant in the dry stage of bronchitis; the bubbling in that of secretion. But both orders of sound are frequently combined in the latter stage; and in some cases secretion occurs so rapidly that bubbling rhonchus is audible from the first. When the secretion is very abundant, distinct agitation of the fluid in the tubes may be caused by the action of the heart. As was first observed by Dr. Stokes, each pulsation of the heart then causes a corresponding rhonchal sound, continuing when the breath is held.

In idiopathic capillary bronchitis, in addition to the signs belonging to bronchitis generally, auscultation discovers fine bubbling rhonchus at both bases posteriorly; coarser rhonchus higher up. If abundant and minute in its bubbles, this rhonchus indicates very positively that the capillary tubes are inflamed; but as fine bubbling sometimes occurs to a limited extent in cases running a mild course, gravitation of fluid from the larger tubes above to the smaller below, is probably sufficient to produce it on a small scale: if confined to one base, or to one or both apices, the bronchitis it depends on is generally either of emphysematous or tuberculous origin.

In intense bronchitis of both lungs, especially where any slight emphysema pre-existed, the bulk of the organs may be sufficiently increased, to push the heart slightly downward, and to the right; the organ then beats mainly at the left costal angle. The diaphragm and subjacent viscera are likewise depressed to a trifling amount.

550. The diagnosis of bronchitis of the large tubes is sufficiently simple,—dry, passing into moist, bronchial rhonchi,

post-sternal soreness, expectoration non-sanguineous, first clear then muco-purulent, accompanied with slight febrile reaction, identifying the affection. It is well to observe here that the mere existence of dry rhonchi is not significant of bronchitis; those rhonchi may doubtless, in emphysematous and asthmatic persons, be produced by spasm of the minor tubes.

551. Capillary bronchitis is distinguished from diffused pneumonia by its clear, or nearly clear, percussion-sound, by the coarseness of its rhonchus, the deficiency of tubular breathing, the comparative coolness of the skin, its less perverted pulse-respiration ratio, the deficiency of rigors of invasion, and its non-sanguineous expectoration.

From the lobular pneumonia of childhood the distinction is less easy. The primary rhonchus of pneumonia in infancy is comparatively coarse, that of capillary bronchitis comparatively fine; and assistance from the sputa fails, as they are almost invariably swallowed. In capillary bronchitis, however, the moist rhonchus is generally much more diffused than in pneumonia, and tubular breathing does not occur; the skin too is free from acrid heat, often temporarily moist.

The diagnosis of capillary bronchitis and acute phthisis is considered with the latter disease.

Typhoid (Peyerian) fever seen within the first few days, before the appearance of its special eruption, might, and actually has been, mistaken for acute bronchitis. But the cough and dyspnœa, which lead in such cases to the error, prove to be excessively slight in proportion to the extent and loudness of the dry, or it may be moist, bronchial rhonchi; the expectoration is sometimes tinged with blood, through co-existent epistaxis; the dull leaden hue of the typhoid facies differs strikingly from its livid tint in severe primary bronchitis. Besides rigors, vomiting, cephalalgia, abdominal pain, common initiatory symptoms in typhoid fever, are almost unknown in bronchitis. In the very rare instances in which they mark the invasion of capillary bronchitis, the whole special symptomatology of the latter disease stands out from the first in such strong relief, as to make error next to impossible.

552. The treatment of acute bronchitis in the adult, sufficiently simple in its general indications, is often beset with difficulty at the bed-side.

Venesection is advisable to the extent of twelve or fourteen ounces, where an extensive surface is involved, where the constitution is strong, and the febrile action positively of sthenic type. Rarely is repetition of general blood-letting called for by the violence of the symptoms; and, while the abstraction of large quantities of blood, with the view of putting an immediate close to the disease, is perfectly chimerical, such sacrifice of the fluid is useless for an object assigned by some writers,—the *prevention* of pneumonia, seeing that in the adult idiopathic inflammation of the tubes does not pass on to the parenchyma. If there be strong apprehension from the past history, or from existing symptoms, of the capillary tubes becoming extensively involved, plausible grounds exist for repeating venesection, especially if the blood previously drawn have proved highly hyperinotic; but it must not be forgotten that, though we may thus control the congestive stage of the capillary disease, there is no evidence of our being able to prevent its occurrence; and the patient will require all attainable strength for the almost inevitable asphyxiating stage. The determination of the amount of blood to be drawn in these cases is the real clinical difficulty in their management; and as, unfortunately, numerical evidence is wanting on the point, the physician must in each case trust for guidance to a careful appreciation of all its circumstances. *Cæteris paribus* venesection may with propriety be pushed further in country than in town-practice. In cases of medium severity, cupping between the scapulæ, to six or eight ounces, or the application of from twelve to twenty leeches to the upper-sternal, infra-clavicular, or axillary regions, will suffice in the way of a first blood-letting,—to be repeated, should the relief to the breathing, as shown especially by the number of respirations per minute, be only temporary. Dry cupping of the chest will sometimes with propriety be substituted in weakly persons on the second and subsequent occasions; or the exhausting apparatus of M. Junod may be applied to one or both lower extremities.

Although not demonstrably so efficient in bronchitis as in pneumonia, tartarised antimony is the most effectual agent known in controlling acute sthenic bronchitis. In divided doses of from four to ten grains in the twenty-four hours, it appears to hasten resolution very sensibly in the milder cases, and advances the secretive stage in the severer. If the tendency to depression under the influence of the antimony be very marked—marked enough to excite fear that the vital powers may be too much lowered—calomel and opium may be employed instead. I entertain no question as to the superiority of tartarised antimony under ordinary circumstances.

Alkalis are theoretically indicated, where the sputa are viscid, and contain exudation-matter; and when the blood is notably hyperinotic. I have, however, little experience of their efficacy in this inflammation.

553. Free secretion from the tubes being established, and the febrile action lowered, the application of a full-sized blister to the sternum, or between the scapulæ (here, in consequence of gravitation, it draws off most fluid), becomes most serviceable;—mustard-poultices may, even from the first, be employed. Diaphoretics and expectorants, containing liquor ammoniæ acetatis, vinum ipecacuanhæ, paregoric, and similar agents, are now advisable,—and these may gradually be replaced by preparations of a more stimulant character, such as tincture of squills, and the ammoniated tincture of opium of the Edinburgh Pharmacopœia. Dilute hydrocyanic acid and the tincture of lobelia inflata, especially if there be spasmodic tendency in the cough, and the fear of serious depression be past, are useful adjuncts. If the abundance and fineness of the moist rhonchi indicate tendency to bronchial accumulation, emetics are of signal service, by mechanically clearing the tubes. The sulphate of

zine, as entailing little or no constitutional depression, is the best agent of the class; and may in urgent cases be repeated thrice in the day. In the advanced stage of the disease, if there be inclination to lapse into a chronic state, carbonate of ammonia, senega, the balsamic medicines, copaiba, gum-ammoniacum, and compound tincture of benzoin, are distinctly serviceable in moderating the amount of secretion, facilitating its discharge, and so relieving dyspnœa.

554. Throughout the whole course of the disease, the bowels should be kept freely open. It would be difficult to prove, it is true, that the duration of the disease is prolonged by confinement of the bowels, but most certainly fulness of the abdomen increases dyspnœa, and discomfort in the chest. On the other hand, I have never seen any utility in severe counter-irritant purgation.

The temperature of the room should range from 63° to 66° Fahrenheit, or even higher under special circumstances, such as previous residence of the patient in a warm climate. The moisture of the atmosphere may be regulated according to the patient's feelings by evaporating water from a dish near the bed; I have known violent paroxysms of cough and dyspnœa relieved by this simple plan. Occasional free ventilation of the room, the patient's head being protected at the time, is essential to his early recovery,—he wants all obtainable oxygen. Flannel should be put on next the skin, if not previously worn.

555. If the disease have actually assumed an asphyxiating character, when the patient is first seen, no matter how that character have been produced,—that is whether by implication of the capillary tubes, by very extensive seizure of the minor ones, or by moderate inflammation occurring in an asthenic state of the system,—depletory measures must be employed with the extremest caution. This is the more true, as it is especially in aged persons the disease is liable to pass suddenly from a very mild state, apparently not requiring medical management, to one of the gravest character. The abstraction

of a few ounces of blood may, under such circumstances, be followed by vital depression, which very manifestly hastens the fatal issue, and is perhaps occasionally its real cause. Local dry cupping, or the application of Junod's apparatus to the limbs, counter-irritation of the chest by mustard poultices and blisters, along with, internally, stimulant expectorants, especially the sesquicarbonate of ammonia, in doses of from three to ten grains every second or third hour, combined with squill, serpentaria and senega or ammoniacum, constitute the staple of the treatment. Muriate of ammonia, in doses of five to twenty grains, is by some preferred to the sesquicarbonate: chlorate of potass, in doses of five to twelve grains every hour, sometimes acts favourably in warding off apparently imminent asphyxia, but on the whole appears to me inferior in efficacy to the latter salt of ammonia. The patient's strength must be supported by strong beef tea and jellies; and wine or brandy given in frequent doses to sustain his calorific power.

In the more serious cases of bronchitis, it is exceedingly probable, the muscular coat of the tubes loses its contractile force. Now that coat appears, in the calm, and still more in the forced expiration, of health, to possess the power of reducing the tubes to less than their medium size;* the elasticity of the inflamed bronchi must likewise be impaired according to one of the acknowledged laws of inflammation. Now thus are easily intelligible the tendency to dilatation of the tubes, the laboured expiration, and the accumulation of secretions, characteristic of the acute disease. And hence, more particularly as bronchial paralysis and air-distension of the lung are known to follow section of the par vagum, it seems right that, as a final resource, galvanism, in the course of that nerve, should be tried in cases of asphyxiating bronchitis.

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* Vide an interesting paper by Dr. Radclyffe Hall, "On the Action of the Muscular Coat of the Bronchial Tubes in Respiration."

II.—CHRONIC BRONCHITIS.

556. Chronic bronchitis, like the acute disease, varies greatly in severity.

557. In one class of cases slight cough, with yellowish-white muco-purulent expectoration, moderate in quantity and easily voided, attended with little or no post-sternal soreness or pain, affecting but very slightly the general health, appetite, and flesh, appearing in winter, and ceasing on the approach of the mild season, constitutes clinically the whole of the disease: this is the slightest form of "winter cough."

558. In a second class of cases the cough is more violent, and more constant, severest in the mornings; the expectoration scanty and adhesive, or easy and copious,—under the latter circumstances consisting of large nummulated masses, floating, semi-sinking, or sinking in water, non-aërated or scarcely aërated, ash-coloured, yellowish-green, deep-green, or in very rare cases of a tint almost like Scheele's green, remaining separate, or forming a single liquid collection, slightly streaked occasionally with blood, when the cough is excessively violent, and especially if the left side of the heart be obstructed in the slightest degree, but never accompanied with actual hæmoptysis,* of peculiar nauseous odour, sometimes fœtid, smelling like wet plaster, or putrescent matter. Microscopically the sputa consist of epithelium, pus-corpuscles, blood-discs, exudation-cells, and exudation-matter in patches, sometimes exhibiting some slight tendency to moulded form. The fœtor described may exist without destruction of substance visible to the naked eye; but it is probable, though I have not actually seen this, that microscopical sloughs may be separated in such cases:

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* "Expectoration of blood, in persons labouring under chronic bronchitis, with or without emphysema, but without notable disease of the heart, justifies in itself a suspicion of the existence of latent tubercles."—Author's Report on Phthisis, as observed at the Brompton Hospital; Brit. and For. Med. Chir. Rev., January, 1849.

gangrenous odor from the secretion of an unbroken surface seems hardly admissible. The varieties of tint are inexplicable by any visible character.

There is no great pain, heat, or soreness in the chest, except after fits of coughing, when it is mainly post-sternal. The respiration ranges scarcely out of proportion with the pulse in frequency,—both being raised, slightly as the habitual state, materially during and shortly after paroxysms of coughing, above the individual standard of health. The appetite fails, the sleep becomes broken, and flesh wastes very perceptibly: I have known as much weight lost during the first three weeks of an annual recurrence of chronic bronchitis, as during the same period in the average of cases of consumption in active progress;—but in bronchitis the weight ceases to diminish after a certain time; in phthisis its diminution, though fluctuating according to laws as yet unestablished, holds on in the main.

If an acute attack or severe exacerbation of the disease occur in a person afflicted with this serious variety of the chronic form, the expectoration becomes in part more viscid, transparent, and highly frothy for a variable number of hours or days; then relapsing into the simply purulent state, grows so excessively abundant as in itself to account partly for the rapid debility that ensues. The laboured respiration, hurried somewhat out of proportion to the pulse, lividity of the lips, malar bones, chin, tip of the nose and finger-ends, coolness of the hands, feet, and it may be, of the extremities generally, clammy perspiration, sometimes rather copious, coolness of the expired air, inability to lie down, sensation of pulmonary oppression and want of air, all point to the asphyxiating character of the attack. Fitful dozes give but temporary relief, and leading to accumulation of secretion, increase the suffering felt on waking. Here is a condition of extreme danger, principally observed in the aged (*senile* bronchitis) and one of the most frequent causes of their death.

559. In a third class of cases, the prominent feature of the disease is a peculiar flux from the bronchi,—whence the name *bronchorrhœa*. In this variety paroxysms of cough and dyspnœa, which may be of almost daily occurrence, or even more frequent, are relieved by copious expectoration of a thin watery fluid, or of a ropy, gluey, transparent substance, like raw white of egg mixed with water,—a quarter of a pint of this may be secreted in the course of half an hour on the decline of a paroxysm. Though sometimes fatal to old people, from their want of power to throw off the accumulated secretion, this form of the disease seems occasionally useful, when slight, in relieving pulmonary congestion dependent on mitral disease, and should not under these circumstances, without mature consideration, be removed—at least completely.

560. There is a fourth variety of chronic bronchitis, to which the rather contradictory name of *dry catarrh* was given by Laennec, characterised by exceedingly troublesome cough, oppression of breathing, tightness of the chest, and sometimes extreme dyspnœa;—expectoration being totally deficient, or consisting of semi-transparent, small, gray, pearl-like, roundish particles. An attack of ordinary bronchitis, with muco-purulent secretion, may occur in such cases, and put a period for a time to the chronic disorder. Pathologically dry catarrh seems to consist in active congestion of the tubes; clinically it is allied to, and often associated with vesicular emphysema.

561. The physical signs of chronic bronchitis are essentially those of the acute disease. There are certain modifications worth attention, however, especially as no single specific sign distinctive of the two forms exists.

Taking the results of a mass of cases, the percussion-sound can scarcely be said, as a rule, to differ from that of health. If on the one hand congestion and thickening of the bronchial walls, coarctation of the tubes, and collapse of lobules or larger portions of lung tend to impair its clearness, on the other the existence of air-distension and emphysema tend to raise the average resonance. Local accumulation of secretion and obstruction sometimes render spots more or less dull one day, which the next recover their clear resonance. But, it must be remembered, that the percussion-sound at the posterior bases, in cases of chronic bronchitis with acute recrudescence, may become markedly dull; and the respiration become high-pitched, and of bronchial or even diffused blowing quality. I have known this state mistaken both for pneumonia and for pleuritic effusion. When, as sometimes happens, one side only is thus impaired in resonance, the error is very easily committed, and probably furnishes a clue to the alleged enormous frequency of fatal pneumonia among the aged in some localities. The persistence of vocal fremitus will habitually distinguish the dulness of bronchitis from that of effusion; the deficiency of true tubular metallic breathing from that of pneumonia. In the latter case, too, the pulse-respiration ratio may be appealed to with confidence; it is never perverted, even in this *accumulative* bronchitis, to the degree that it is in pneumonia: besides, though the vocal resonance may be strongly bronchophonic, it is not tubular, sniffling, and metallic.

Chronic bronchitis tends to distend the lungs *generally* (though, from bronchial obstruction in some spots, it may have the reverse effect on them *locally*), and hence to widen the chest, and even depress somewhat the heart and diaphragm. But it is very difficult to demonstrate to what amount bronchitis is capable of carrying these changes; it is so constantly associated with their more active and positive cause—emphysema.

The respiration-sounds vary in force and quality widely in different parts of the same lung. The quantity of the respiration falls on the whole greatly below the average; harsh and coarse, the loud inspiratory and expiratory sounds audible in some spots differ wholly in character from those of true exaggerated respiration, are probably produced in the capillary and larger tubes, and resemble dry rhonchi in properties (sonoro-sibilant respiration). Full respiration will sometimes be heard after free expectoration, in a spot where a moment before none, or next to none, was audible. True blowing respiration is never heard in bronchitis. The rhonchi are those of the acute disease; the vocal resonance very feeble, or strongly bronchophonic.

562. Anatomically the disease is characterised by congestion, and widening of the vessels, unevenness of the mucous membrane and epithelial abrasion; the walls of the tubes are thickened and hard, from infiltrated induration-matter, which sometimes extends beyond them, and encroaching on the adjacent lung-substance, obliterates its capillary vessels; the pressure of this matter diminishes the calibre of the tubes both directly and indirectly by circular constriction; and actual obliteration of the finer bronchi may occur, where in addition, exudation-matter has hardened in patches on their internal surface. Neither ulceration nor sphacelus, visible to the naked eye, belong to simple chronic bronchitis. The longitudinal and circular muscular fibres are sometimes much hypertrophied. The cartilages of the larger tubes occasionally calcify.

563. In the acute disease the bronchi may, as we have seen, undergo dilatation, the more readily, the younger the individual. There is every reason to believe that, with the recovery of the tone of the bronchial muscles, the tubes gradually resume their natural form. I have seen cases of intense bronchitis in childhood, lapsing into the chronic state, and attended with signs apparently depending on widening of the tubes, which signs have gradually and totally disappeared. So likewise in the chronic disease, the bronchi sometimes dilate; but, once established, the dilatation remains a permanent evil. Several tubes commonly undergo pretty uniform dilatation, resembling the distended fingers of a glove; in other cases one or more bronchi become globularly dilated in one or more points. Beyond dilatations of the latter kind, each tube may be

excessively reduced in calibre, or actually obliterated.* The surrounding lung-substance, condensed by pressure, consolidated by chronic pneumonia, or by infiltrated induration-matter, is more or less extensively impermeable to air.

564. The physical signs are peculiar. The bulk of a lung having several of its bronchi dilated, falls below the average, especially in the neighbourhood of the widened tubes: this comes of the co-existing condensation of lung-substance. Hence it is, that instead of any tendency to local bulging, there may be distinct depression of the corresponding chest-surface. And in those cases of dilated bronchi, where the intra-parenchymatous and intra-lobular cellular tissue of the lung is infiltrated extensively with induration-matter (Corrigan's cirrhosis), the affected side generally may be the seat of marked retraction.† Generally speaking, the vocal fremitus is increased in intensity, and may be greatly so; rhonchal fremitus, likewise, is sometimes very strong.

I have never known dilatation of the tubes, or even of a single tube, to any notable extent, unattended either with dulness under percussion, commensurate with the extent of attendant condensation, or with some form of tubular resonance. In certain published cases, where the resonance is alleged to have been "clear," the so-called clearness was doubtless of the latter unnatural quality. I have known it very purely amphoric.

The respiratory murmurs are simply harsh, or high-pitched and bronchial, or, more commonly, of diffused blowing type. When one or more tubes are largely dilated, the respiration may be clear, ringing and distinctly cavernous in quality; and, under the same circumstances, the ordinary dry or moist bronchial rhonchi may be superseded by the dry or moist cavernous varieties. The vocal resonance varies: totally deficient in some cases, temporarily, or, to all appearances, persistently; in others it is strongly bronchophonic, and may even assume pectoriloquous quality.* The heart's sounds may be transmitted with undue intensity through the seat of dilatation.

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* Osmond, U. C. H., Males, vol. iv., p. 343.

† Osmond, U. C. H., Males, vol. iv., p. 341, &c. Here the retracted lung weighed 55½ oz., but it was also tuberculised.

565. The addition of dilatation of the tubes to chronic inflammation of their mucous membrane, seriously increases the gravity of the latter disease. Here it is that the expectoration is most abundant, most opaque and solid, most thoroughly purulent, and hence most wasting to the system. The aëration of the blood is rendered difficult by the altered structure of the mucous membrane; hence lividity of the face commonly exists to a marked amount. The long continuance of the disease tends to produce enlargement and thickening of the right ventricle, but I have never observed hæmoptysis unless where there was co-existent mitral disease or pulmonary tubercle. Some degree of night-sweating occasionally occurs; and the weight of the individual falls very considerably below the standard of health.

566. This symptomatic state, far from dissimilar to that of phthisis, may coexist with physical signs, so like those of excavation, that it is next to impossible sometimes to affirm with certainty whether a given case be one of tubercle with cavity or cavities, or of globularly dilated bronchus or bronchi with surrounding induration.† The distinction may, however, in the majority of instances, be established by means of the following points. The percussion-sound is invariably dull above the clavicle in cases of phthisis of the ordinary class; not necessarily so in those of dilated bronchi: hence clear resonance in this spot will argue strongly for the latter disease; but dulness of sound will not decide the point against it. Generally speaking, too, the dulness is more absolute and

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* Osmond, U. C. H., Males, vol. iv., p. 341, &c.

† Case lxi. of Louis, Phthisie, 2ème éd., p. 562, is sufficient proof of this.

extensive below the clavicle in phthisis than in the other affection; for, if the bronchi be largely dilated and surrounded with much induration-matter, the resonance becomes tubular or amphoric. The signs of tuberculous excavation are found at the apex: those of dilated bronchus generally lower,—say at the union of the upper with the middle third of the chest. When tubercle has reached the excavation-stage, flattening of the infra-clavicular region is, relatively to that of the side generally, more marked than in bronchial dilatation. I have never known hæmoptysis produced by chronic bronchitis with dilatation alone;—if hæmoptysis exist, and there be no evidence of mitral disease, the inference that the excavation is tuberculous becomes matter of necessity. Extreme emaciation, profuse night-sweats, and obstinate diarrhœa, do not, as far as I have seen, come of the bronchial disease alone: all three may, however, be wanting in phthisis. The course of the physical signs will avail us also, if the case continue for a time under observation. In phthisis the signs are, as a rule, constantly increasing in degree and extent; in bronchial dilatation, they may remain for months unaltered in both these respects: dulness under percussion, as remarked by Dr. Stokes, precedes the signs of cavity in phthisis, and does not occur till after them in bronchial dilatation;—to the latter clause, however, I have seen exceptions.

567. I once met with a case* where the conditions of bronchial dilatation were sufficiently defined to justify the diagnosis of that state,—and where, after death, both bronchial dilatation and a small recent growing tuberculous excavation were found almost side by side. This compound state is, probably, beyond the reach of diagnosis.

And, in point of fact, the association of dilated bronchi and tuberculisation of the lung is by no means uncommon, due admission being made for the rarity, absolutely speaking, of the former. Tuberculous excavations and gray granulations may exist in the same lobe with globularly dilated bronchi;* or the former may affect the upper, the latter pervade the lower, lobe.† Under these circumstances the dilatatious might readily be be taken for smooth-walled excavations, did not the hypertrophous muscular fibres prevent the error.

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* Ann Harrison, U. C. H., Jan. 22, 1848.

568. In the treatment of chronic bronchitis, whether accompanied or not with dilatation of the tubes, it is very rarely advisable to take blood from the arm, even during acute exacerbations;—the strength fails rapidly in such cases from loss of blood; and to bleeding at the outset, rapid asphyxia at the close, from inability to expectorate, may often be traced. A few ounces, say, in ordinary cases, about four or five, taken locally by leeching or cupping, are as much as may be abstracted without fear. Free dry cupping, with flying blisters applied occasionally to different parts of the chest, are among the most effectual means of counteracting the ill effects of acute exacerbations. In the purely chronic state, counter-irritation, with tartar emetic, croton oil, or the turpentine and strong acetic acid liniments, is essential in the treatment, unless emaciation be very far advanced, or the skin peculiarly irritable. The inhalation of tar vapour, creasote vapour, iodine, or chlorine, most unquestionably reduces the irritability of the mucous membrane, and the quantity of secretion; the results of M. Cottereau, with chlorine inhalations, are peculiarly important, and show, as admitted by M. Louis, that singular advantage may be obtained through them, even where the general symptoms closely simulate those of phthisis. Of the singularly beneficial effects of creasote vapour, in particular, I can speak with confidence; the cases are rare where it fails to agree from the first; but in very irritable constitutions the mucous membrane may gradually be prepared for it by the

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* Osmond, U. C. H., loc. cit. p. 343.

† Maddox, Males, U. C. H., vol. ix., p. 240.

inhalation of extract of hyoscyamus, or conium. If there be spasmodic tendency, the latter may be rendered powerfully antispasmodic and sedative by the addition of a few drops of liquor potassæ.

The choice of internal remedies will vary with the condition of the discharge from the tubes, and the amount of general re-action. If there be little expectoration, an excited circulation, and a tendency to congestion of the parenchyma, tartar emetic in small doses, ipecacuanha, or colchicum, are the best agents, combined variously with hydrocyanic acid, lobelia inflata, belladonna, stramonium, hyoscyamus, paregoric or similar drugs. In a few cases I have seen mercury very rapidly lower the circulation, and induce free discharge. If there be but little vascular excitement, squill, senega, ammoniacum, with opium, are preferable combinations. Balsam copaiba and the compound tincture of benzoin may be joined to these stimulants,—the former especially seems to exercise a specific soothing effect on the mucous membrane. Muriate of ammonia is strongly recommended by some writers. Medicinal naphtha will control superabundant discharge. Iodide of potassium, in sufficient doses to produce slight iodism, occasionally relieves the dyspnœa and chest-oppression to a very striking degree;—in such cases does it promote absorption of exudation-matter? From ordinary alkalies I have seen no positive benefit, except where the individual was of rheumatic or gouty diathesis; and then the accompanying bronchitis was not, properly speaking, of the simple variety now under consideration.

The jellies made from Iceland and Carragheen mosses both soothe the cough and afford nourishment. If emaciation occur, cod-liver oil should decidedly be employed;—many of the good effects of the oil, as observed in phthisis, are yet more readily produced in chronic bronchitis. Bark and the mineral acids are useful in controlling debility, and improving the appetite: if anæmia supervene, iron becomes essential.

The diet should be nutritious and non-stimulant. Where circumstances permit, change of climate should be tried, either for the winter or permanently (see Appendix); where this is impossible, a steady temperature of 60° to 64° Fahr. should be maintained in-doors; and the mouth and nose protected out of doors by a respirator.

569. During the paroxysm of *bronchorrhœa*, the hot bath, or the hot air-bath, sinapisms to the extremities, emetics, full doses of lobelia inflata, and, if there be failure of vital power, sesquicarbonate of ammonia and brandy, are the chief remedies.

570. In the treatment of an acute attack, supervening on the chronic disease in a person of advanced years, the caution already given as to blood-letting in simple acute asphyxiating bronchitis, seems to me of yet greater importance. Here it is not the inflammation that kills; it is the vast accumulation of muco-purulent secretion supplied by a congested surface,—secretion which prevents oxygenation of the blood, and which the strength of the patient fails to throw off—that really destroys life: the brain and tissues become poisoned, too, with venous blood. Sesquicarbonate of ammonia is, in such cases, required almost from the first: it is best given in combination with squill and nitric ether. If any sinking tendency appear, chloric ether acts as a more powerful general stimulus in these cases than any medicine I am acquainted with. Nux vomica and galvanism are worthy of trial as direct excitants of the muscular coat of the bronchial tubes; and congestion might be relieved by the application of Junod's apparatus to the lower extremities.

In cases of this class terminating fatally, the result is commonly held to be immediately brought about by pneumonia: a notion derived sometimes from the occurrence of dulness under percussion at one or both bases,—sometimes from *post-mortem* examination,—sometimes from both sources. I have already pointed out the source of fallacy in the percussion-dulness referred to; and I have great doubts of certain consolidations found after death (peri-pneumonia *notha* in more senses than one) being truly pneumonic. It has not occurred to me to meet with such consolidation, except where there was co-existent heart-disease, and more especially of the mitral orifice,—consolidation hence mechanically, and not actively congestive.

III.—VARIETIES OF BRONCHITIS.

571. The varieties of bronchitis may be classified as follows:—

Varieties dependent on—	
1. Period of life	Bronchitis at birth; infantile; adult; senile.
2. Nature of secretion	Dry; serous; muco-purulent; plastic.
3. Nature of cause	Mechanical bronchitis; hay-asthma.
4. Amount of prevalence	Sporadic; epidemic (influenza).
5. Association of spasm	Hooping cough; asthmatic bronchitis.
6. Secondary origin	Secondary to— General diseases. Typhoid and typhus fevers; the exanthemata. Blood-diseases. Syphilis; gout; rheumatism; Bright's disease; glycohæmia;* scrofula; jaundice; scurvy; purpura; cancer. Local thoracic diseases. Tubercle, cancer, chronic inflammation of the lung; heart disease; aneurism of the aorta, &c.

572. The peculiarities of some of these varieties have already been referred to; a few of the others require special consideration.

PLASTIC BRONCHITIS.

573. We have already seen that plastic exudation-matter forms to a limited extent in simple bronchitis; the profuse production of such matter distinguishes one variety of the disease, which is probably, though not demonstrably, dependent on a peculiar diathesis.

574. An affection of great rarity, true plastic bronchitis is anatomically characterised by the formation of solid or tubular concretions of exudation-matter of low type † within the

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* Suggested (from γλυκυς—ἅιμα) as a substitute for "saccharine diabetes."

† I have found them to contain exudation-cells; some nucleated, the majority not so. In the main, the substance is fibrillar or amorphous.

bronchial tubes, reaching, more or less extensively, from their finest to their largest divisions. The disease has little tendency to spread upwards: the trachea remains unaffected; the voice, though sometimes becoming slightly husky, habitually retains its natural quality and strength. On the other hand, plastic inflammation extends downwards from the larynx to the bronchi in a small proportion of cases of croup,—but with these cases we have nothing to do here.

575. Clinically plastic bronchitis is distinguished by its chronicity, and frequent acute recrudescences, and its comparatively slight influence on the general health. The physical signs are also peculiar; disappearance of all respiratory sounds in given spots of the lung from time to time, marks complete obstruction of a large communicating bronchus,—and dulness, as complete as in pneumonic consolidation, probably from collapse of the lung-substance, may occur co-extensively with the deficiency of respiration.* Local pneumonia, attended with pain, rusty, viscid expectoration, true crepitant rhonchus, and blowing respiration, also occasionally occurs in these cases,—generally speaking, running its course uninfluenced, at least perceptibly, by ordinary treatment. Where a very large tube chances to be blocked up, asphyxia may be temporarily threatened; † and oppression of breathing, disproportionate to the apparent amount of disease, is always a prominent symptom.

The expectoration of the casts is generally preceded by some hours' dyspnœa and hacking dry cough; and during the periods of acute attack, I have found the pulse-respiration ratio vary from 2·2:1 to 3·5:1. During these attacks, casts of notable size are brought up, generally from three to six or seven times a week,—but small fragments are much more frequently expectorated. Unless the sputa be closely examined under water, particles of concretion escape notice amid the viscid mucus with which they are generally associated.

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* Moss, U. C. H., Females, vol. i., p. 187 (1846), and vol. iii., p. 83 (1848). Expectoration of casts of the tubes commenced, in this case, in the spring of 1843, and, with occasional intermission, continued to the autumn of 1850, when the patient was last seen.

† U. C. Museum, No. 2124.

Streaks of blood, either on the external surface of the casts, or, more rarely, on their internal surface, if they be tubular, are not uncommonly seen; and spitting of florid blood in streaks, or even in drops, mixed with mucus, for a short while after their expectoration, occasionally occurs. This is especially the fact at the height of the acute attack. As this wears off, the concretions and the expectoration become bloodless. Cases have, besides, been observed, in which copious hæmoptysis had occurred for some time previous to the expectoration of solid casts of the tubes. The nature of these cases, however, requires further investigation. It appears very unlikely that they belong to the same class as true plastic bronchitis; the concretions are most probably simple fibrinous coagula from hæmoptoic blood, itself the result of tuberculous disease. In no case of the kind that I have read the record of, was the absence of tuberculous disease proved; and, on the other hand, the occurrence of moulded coagula in tuberculous hæmoptysis, though, for obvious reasons, rare, is sometimes (I have seen it myself) positively observed.

576. During the period of acute seizures the treatment is to be conducted on the same principles, as if the secretion-products were of the ordinary kind. I have not seen any benefit derived from mercurial action on the system. The young practitioner must not confound the local dulness under percussion, which may come on in a few hours in these cases, and depends on obstruction of tubes, with true pneumonic loss of resonance. The weak or suppressed respiration of the former condition, the tubular blowing of the latter, will distinguish the cases.

Few affections of the lungs are more difficult to cure permanently than this. Theory leads to the use of iodine by inhalation and otherwise, and suggests a prolonged trial of alkalies, as diminishing the tendency to hypcrinosis. I have perseveringly employed these remedies without any permanent effect on the disease. It disappears for a time, to return again without obvious cause,—a mode of progress plausibly referrible to the influence of a persistent diathesis.

MECHANICAL BRONCHITIS.

577. Under this head fall those well-marked varieties of bronchitis induced by the inhalation of irritating particles of various kinds. The knife-grinders' rot is primarily mere bronchitis produced by the entry into the tubes of metallic particles and gritty dust from the grinding-stones; miners (whether coal or other), not, as was once erroneously supposed, from the inhalation of coal-dust, but really from that of the soot of the oil-lamps used in working, where the safety-lamp is not employed, are subject to a similar disease; so, too, are quarry-men, cotton-batters, &c. In all these cases the disease (grinders' rot, black phthisis, stone-phthisis, cotton-phthisis, &c.), is essentially bronchitis at the outset, which becomes chronic, is followed by dilatation of the tubes, and, eventually, inflammatory destruction and excavation of the lung-substance itself. But there is no connection between this destruction and the presence of tubercle,—which, if it exist, is purely accidental.

578. The physical signs are those of bronchitis, dilated bronchi, occasionally of emphysema, of consolidation, and, finally, of excavation. The general symptoms are not proportional in severity to the local disease; whence a distinction between these affections and phthisis. The knowledge of the cause also aids, in the diagnosis,—though, on the other hand, it may tend to deceive, as of course these artisans are not exempted from consumption.

579. Cure is impossible, unless the patient change his occupation. Various mechanical contrivances have been invented for the prevention of these diseases, especially among knife-grinders. Abraham's magnetic mouthpiece attracts metallic particles, but has no effect on the stone-grit. Dr. C. Holland's

revolving fan, acting by a strong current upon the spot where the metallic dust and stone-grit are formed, seems to be more successful.

HAY-ASTHMA.

580. A singular variety of naso-pulmonary catarrh, which has been supposed to follow the inhalation of the aroma of the sweet-smelling spring grass and hay (anthoxanthum odoratum) is known under the name of hay-asthma, hay-fever, or summer catarrh. The complaint occurs only at the periods of hay-making, or when the odour of grass is powerful; and is of exceedingly rare occurrence. The susceptibility to these emanations, indeed, constitutes a very remarkable example of unalterable idiosyncracy. Persons who have once suffered, invariably have a return of the disease, if exposed even in a slight degree to the specific cause.

Besides, the perfumes of various flowers and the powder of ipecacuanha produce, in persons peculiarly constituted, symptoms very closely similar.

581. It is scarcely correct to term the disease bronchitis; the entire naso-pulmonary tract is, in truth, implicated. Mid-frontal cephalalgia, violent and continued sneezing, irritation in the eyes and nostrils, with flux; soreness and prickling sensations in the throat; dyspnœa, sometimes paroxysmally severe; post-sternal oppression and rawness; dry, harassing, cough; and, towards the close of a seizure, thin mucous and watery expectoration, are the symptoms of the complaint.

If the affection be left to itself, its usual duration ranges from two to five weeks; but even in cases carefully tended, the symptoms may persist for a month. The annual return of the disease is one of its essential characters.

582. The most effectual means the habitual sufferer can command of preventing an attack, is by removing at the season to the sea-side,—by getting out of the reach of the odours of grass and hay. But so exquisitely sensitive to such sensations

are some individuals, that a land-wind, blowing for a few hours only, will bring on an attack even at the sea-shore. Once the complaint is established, total abstraction of the existing cause will not put an immediate term to the seizure; I have had a very precise narrative of a case, in which the patient retained his symptoms during a passage across the Atlantic.

Several years since, a course of sulphate of iron and quinine and the use of the shower-bath, directed by Dr. Gordon,* removed the complaint in two persons who had been its annual victims for fifteen or twenty years. During the seizure, small, frequently repeated doses of prussic acid, and the æthereal tincture of lobelia inflata are valuable remedies; there seems, in truth, to be some spasmodic element in the disease. Dr. Elliotson believes he has observed benefit felt from breathing an atmosphere with chlorine diffused through it,—saucers of the chlorides being placed in the rooms of the patient's house, and the face washed with a weak chlorinated solution. I am disposed strongly to recommend a trial of creasote-inhalations, once or twice daily, from their remarkably satisfactory effects in two instances.

Are there any drugs having specific influence on the complaint? Dr. Watson recommends arsenic; Mr. Hamerton and Dr. Gream nux vomica. I do not question the occasional utility of either: but I have known arsenic fail egregiously, where it was administered under circumstances peculiarly favourable to fair trial; and I am informed that nux vomica cannot be depended upon. There can be no question that all general tonic measures calculated to invigorate the system, and lessen the special susceptibility of the mucous membranes, as advised by Dr. Gordon, are rationally indicated. They will, however, prove wholly incapable, in a good proportion of cases, of averting the returns of the disease.

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* Medical Gazette, vol. iv.

EPIDEMIC BRONCHITIS, OR INFLUENZA.

583. Influenza, or epidemic catarrh, though claiming a place more naturally among bronchitic diseases than any others of the respiratory passages, is evidently, even in its local manifestations, an affection *sui generis*, and of much more extensive seat, even anatomically speaking, than its classification with bronchitis would seem to signify. Still, in the majority of cases, the most prominent local symptoms are those of bronchitis.

584. Essentially constituted by catarrh, with nervous and muscular prostration, influenza sets in with lassitude, chill, rarely amounting to actual rigors, and aching pains in the limbs; and more frequently, perhaps, affects the mucous membrane of the throat at the outset than that of the nose or air-passages. So, too, nausea and vomiting may precede all other symptoms, showing that the surface of the upper part of the alimentary canal early participates in the disease.

585. Heat and dryness of skin; frontal headache, sometimes excessively severe, particularly opposite the sinuses; small, weak, and frequent pulse; nasal flux, watery, saline and profuse; in rare cases epistaxis; cough of variable severity; expectoration scanty and pituitous; slight dyspnœa; uneasy pain behind the sternum; intercostal neuralgia; tenderness under pressure and sensation of rawness at the epigastrium; white furred tongue; nausea, with absolute anorexia, and occasionally vomiting; diarrhœa of trifling amount; vertigo; tinnitus aurium; pains in the neck, scalp, and over the malar bones; extreme general uneasiness; aching pains and soreness of the limbs and trunk,—all combined with excessive depression of spirits, and an amount of debility and prostration totally out of proportion with the local ailments (syncope sometimes occurs in the erect posture)—are the symptoms of the established disease in its ordinary and pure form.

The physical signs are those of bronchitis, or there are none of positive character; I have noticed this deficiency of notable bronchial signs, even where cough and post-sternal pain existed to a very appreciable amount.

586. There are exceptional cases where violent headache, flushing of the face, and delirium, with fever, are the prominent symptoms; others, where the digestive organs alone suffer, and in the upper parts; others marked by diarrhœa or pseudo-dysentery; and yet others where, erethism or actual catarrhal inflammation of the urinary organs is the main phenomenon. In some cases rheumatic periosteitis of the bones of the face, with considerable tenderness under pressure, continues for days after the other symptoms have yielded. In certain epidemics, the substance of the lungs has been very commonly attacked, and pneumonia cut off multitudes of aged and debilitated persons. Pleurisy is also an occasional complication. Both affections are habitually latent, and to be discovered by their physical signs alone.

587. The nature of the atmospheric poison generating influenza is yet a mystery. But whether constituted by ozone or not, and no matter what accidental peculiarities of course and symptoms the disease may exhibit in particular instances, there is reason to believe the poison is always essentially one and the same. Influenza is, according to all attainable evidence, non-contagious.

588. Terminating, in ordinary cases, by diaphoresis, sometimes by, or rather with, cutaneous eruptions, at the end of from three or four days to a week, influenza invariably leaves, as its sequence, more or less enduring debility, and, in many cases, chronic bronchitic cough. That the symptoms of phthisis have occasionally first become apparent after an attack of influenza, is unquestionable; and the fair inference is that it accelerated the outbreak of the tuberculous disease. Influenza rarely kills those it attacks, unless, aged and debilitated, they have already one foot in the grave. Among this class of the population, the

mortality occasioned by an epidemic has sometimes proved extremely serious.

589. The experience of centuries has established, beyond question, the impropriety of depletory measures in the treatment of influenza. The Czar of Russia might at least plead, in justification of his ukase against blood-letting, the mortality clearly traceable, not only in his own dominions, but in various countries of Europe, England, France, Spain, Italy, to the practice. I doubt if the occurrence of consolidation-signs even warrants the use of the lancet: such signs are observed chiefly in the aged and exhausted; and I know, from experience, that dry-cupping and sesqui-carbonate of ammonia will remove these signs in such cases; whereas I have never seen any benefit derived from abstraction of blood, either by leeches or cups. Such consolidation is very positively *passively congestive* in the aged and infirm, and is increased by depressing measures. Should well-marked signs of sthenic pneumonia occur in a young or middle-aged person, leeching or cupping may be advisable:—but even then caution is requisite; a dozen leeches have, within my experience, produced successive fainting fits in a previously healthy and robust individual. I have never seen a case in London where venesection seemed advisable; perhaps such may occasionally be met with among the more vigorous inhabitants of rural districts. Neither should tartar emetic, active purgatives, nor (I think, in spite of the encomiums of Dr. J. Davies) mercury, with a view to its constitutional action, be employed.

The treatment I have found most successful, is as follows:—Keep the patient in bed; open the bowels by some gentle laxative; give diaphoretics in combination, especially if there be rheumatic pains, with colchicum and an alkali; procure sleep by extract of lettuce, or of hyoscyamus; and allow diluents freely. After the first three or four days, if bronchitic rhonchi exist, a blister should be applied between the shoulder-blades or to the sternum, and an expectorant mixture prescribed. This mixture may with advantage be made somewhat stimulant;—let the vehicle, for example, be partly ammoniacum, serpentaria, or decoction of senega; lobelia inflata, in small doses, and paregoric should enter into its composition. Tonics, iron, and quinine may be given daily during convalescence, unless the stomach have been implicated to any amount. If there be much exhaustion from the first, sesquicarbonate of ammonia and strong beef-tea should be given without hesitation; and the free use of wine or brandy may eventually become imperative.

HOOPING-COUGH.

590. Hooping-cough, or pertussis, seems practically composed of reflex spasm of the air tubes, and special bronchitic irritation, the speciality of character being indicated by the nature of the secreted product.

591. Three stages of the disease are generally recognised: the catarrhal; the spasmodic; the terminal.

592. The catarrhal stage, lasting from one to two weeks, is marked by coryza, general irritation of the air-passages, dry, or almost dry, and slightly paroxysmal, cough, and feverish action. If there be expectoration, it is in nowise specific in character.

593. The commencement of the spasmodic stage is announced by the distinctive, abrupt paroxysms of cough, occurring at irregular intervals by day and night; during these fits both inspiration and expiration are, in their several ways, laborious,—the former prolonged, and accompanied with a loud, cooing noise, called hooping, and obviously caused by partial closure of the glottis; the latter consisting of a number of successive forcible puffs, without obvious intervening inspiration, performed with almost convulsive energy. These successive expiratory efforts seem to force all the supplementary and some of the residual volumes of air from the lungs [82], and it is often not until some of the minor phenomena of asphyxia become apparent,

that the patient gets momentary relief, by the prolonged cooing inspiration. A complete paroxysm, consisting of a variable number of these inspiratory and expiratory efforts, may last from less than a minute to a quarter of an hour or upwards: when thus prolonged, not only do the face and eyes become painfully turgid and livid, but blood may issue from the mouth, ears, and nose; the conjunctivæ become ecchymosed; slight convulsions occur; and involuntary discharge of the fæces and urine may take place,—probably rather of expulsive than paralytic mechanism. The pulse is frequent, greatly out of proportion to the number of inspirations; but it becomes natural, unless some complication exist, after the close of the fit. The paroxysm terminates commonly by expectoration, (during the early part of this stage, thin, scanty, and pituitous; during its more advanced part, ropy, semi-transparent, albumen-like); or by vomiting;—or by pure exhaustion, without discharge of any kind. And in a few minutes, sometimes a few seconds, the patient feels as in health, except from some slight sensation of fatigue, and, if a child, resumes his play, as though nothing had happened. The frequency of recurrence of the paroxysms varies greatly; there may be but two in a day, or two or three per hour. Sometimes ensuing without any immediate excitation, they are more frequently traceable to such influences as irritate the excito-motory system,—the act of swallowing, sudden draughts of cold air, a fit of anger, abrupt movements of the body; like spasms of reflex mechanism generally, they often come on by night. The ordinary duration of this stage ranges from six to eight weeks; but it may terminate by the third week, or continue for months.

594. During the terminal stage, the cough becomes less frequent, loses the convulsive and hooping characters; the expectoration ceases to wear the albumen-like, ropy appearance, and becomes simply catarrhal. Eventually all symptoms disappear in the course of from one to three weeks.

595. In a certain number of cases small, round, superficial

ulcerations form at the root of the tongue; hence the disease has been fancifully assimilated to hydrophobia, an affection reflex-spasmodic in character, and attended with vesicle-formation at the root of the tongue.*

596. The physical signs are only negatively important; they exclude diseases which might erroneously be supposed to be present. During prolonged paroxysms, and especially towards their close, the percussion-sound falls somewhat in clearness: I state this from positive observation; but it is no more than might be expected from the forced evacuation of the air-cells that occurs. During the brief efforts at inspiration between the successive expiratory puffs of the cough, inspiratory murmur may sometimes be caught; but during the noisy and hooping inspiration, very little true breathing-sound reaches the surface; probably spasm of the bronchial tubes prevents the air from advancing to the vesicles. Sonorous and sibilant rhonchi, and, if there be much fluid in the tubes, the varieties of the moist bronchial rhonchi, are heard.

597. Hooping-cough is in some cases an affection so mild, and its special characters so feebly developed, that doubts may be entertained as to its having existed at all; in others so severe as to destroy life. Death has occurred from asphyxia, from rupture of the air-cells, and effusion of air into the mediastinum and common cellular membrane, or from rupture of the pleura and pneumothorax, or from the mere exhaustion of the protracted disease. Various of its complications, capillary bronchitis, pneumonia, croup, or convulsions, also occasionally prove fatal.

598. In mild cases of hooping-cough little is required in the way of treatment. Attention to the state of the bowels, the use of gently soothing expectorants, limitation in eating, warm clothing, and avoidance of all the *lædantia*, are all that is called for.

599. In some cases during the catarrhal stage, purgatives and

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* Lersch, in Journ. für Kinderkrankheiten, 1844.

antimony, or ipecacuanha, in doses measured by the patient's age, are desirable. Unless the symptoms are sharply inflammatory, blood-letting in any of its forms should be avoided; the disease must run a certain course, and the patient's strength requires husbanding.

Various means of lessening the severity of the paroxysms may be had recourse to. Nauseants, given also occasionally in emetic doses, are among the best of these,—antimony, ipecacuanha, and lobelia inflata, especially the two latter, in combination. Antispasmodics, such as asafœtida (if unbearable by the stomach, rubbed over the epigastrium and spine), musk, valerian, and camphor, answer well in some cases. Of narcotics, hyoscyamus, lettuce, and conium are the safest; opium should not be given unless in combination with ipecacuanha. Belladonna, pushed to the verge of poisonous effects, is sometimes a justifiable remedy, where the paroxysms are dangerous *per se*; but I have no evidence that it shortens the disease. Hydrocyanic acid, as a rule, is a fitter agent; and may be trusted to throughout as the most direct sedative of the spinal cord: it seems sometimes to bring the duration of the complaint much within the mean.* Chloroform, in doses of from three to ten minims according to age, lessens the severity of the fits.

Nervine tonics, such as oxide of zinc, nitrate of silver, and iron, are sometimes required before the close of the disease.

Counter-irritation by tartar emetic, croton oil, or acetic acid and turpentine, is decidedly useful: such applications should be used of low strength. A blister kept on for two or three hours, and followed by a linseed poultice, sometimes greatly lessens irritation for several days. Belladonna-frictions to the spine, or a broad strip of belladonna plaster, extended from the nape of the neck to the loins, are distinctly serviceable in many cases. Morphia, applied endermically to the throat, is favourably spoken of. A fit may sometimes be shortened by a draught of cold water, or by the cold-water-dash to the face. The shower-bath, in the case of children, generally does more harm than good.

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* A. B. Granville, Treatise on Hydrocyanic Acid. London, 1820.

Chloroform-inhalation may be had recourse to in bad cases: of its influence in shortening the paroxysm, and weakening the force of the spasm, there can be no doubt. The quantity inhaled should be just sufficient to produce slight cutaneous insensibility, never to bring on narcotism.

Touching the orifice of the glottis with a strong solution of nitrate of silver seems to lessen the irritability of that part.

Of various alleged specifics, such as sulphuret of potassium, cochineal, nux vomica, arsenic and cantharides, nothing satisfactory can be said. Alum, in small and repeated doses, extensively tried by Mr. W. B. Hutchinson at the Foundling Hospital, appears to him, though wanting in the specific virtues announced, to shorten somewhat the average duration of the disease, and render the paroxysms less violent. The disease never, according to Dr. Arnoldi, resists longer than three weeks the influence of nitric acid, diluted to the acidity of lemon juice, freely used: a child, aged two years, took in this form one and a half drachms of concentrated nitric acid in the day with the best effects.

During the first and second dentition, the teeth should be carefully looked after. Flannel should be worn next the skin, a uniform temperature maintained about the patient as far as possible; and, if the complaint set in towards winter, and removal to a warm locality be impossible, he should be confined to the house absolutely. Towards the close of the disease change of air, as in the somewhat analogous case of laryngismus stridulus, proves of signal benefit; even at the outset I have known mere change of the kind, without any superiority of atmospheric conditions, favourably modify the complaint.

600. Strong evidence of the transmissibility of hooping-cough from individual to individual is from time to time met with. Dr. Neil Arnott has related to me the following remarkable

illustration: a lady left a port in the East for England with two children in the hooping-cough; the vessel put in at St. Helena, where the linen of the children was sent ashore to be washed; the children of the laundress were seized with the disease; and from them it spread generally through the island,—no case of the complaint having previously occurred there for a lengthened period. Seclusion is, therefore, advisable.

BRONCHITIS SECONDARY TO GENERAL DISEASES.

601. Symptoms of bronchitic character occur in the great majority of cases of continued fever of "typhoid" type,—that is, characterised by alteration of the glands of Peyer.

602. Commonly unattended, as it is, with post-sternal or other pain, or accelerated breathing, and causing little cough and little or no expectoration, generally-diffused dry and moist bronchial rhonchi, especially the former, are the sole evidences of inflammatory affection of the tubes. The existing inflammation is evidently very slight. The disproportion between the amount of rhonchi on the one hand, and of the cough and dyspnœa on the other, might, according to M. Louis * be fairly used as an element of diagnosis, and argue for the secondary character of the existing bronchitis.

603. In cases of this class the state of the bronchi is unimportant in regard of treatment. But in comparatively rare cases severe bronchitis, with extensively diffused fine bubbling rhonchus, accelerated breathing, and free expectoration, occurs, throwing into the shade the more ordinary characters of the fever. Counter-irritation and soothing, or eventually stimulant, expectorants are, however, solely required even in this severer class of bronchitic cases. Singularly enough, there is no special tendency to accumulation of secretion in the tubes.

604. The precise researches of Dr. Jenner show that there is less tendency to bronchitis in the "typhus," and still less in the "relapsing," than in the typhoid, types of continued fever.

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* Affection Typhöide, vol. ii. p. 283. 1ère édit.

BRONCHITIS SECONDARY TO BLOOD DISEASES.

605. Bronchitis dependent on blood-diseases is characterised s a rule by its pertinacity, and its resistance to ordinary ntiphlogistic treatment. The measures adapted for its relief and cure are substantially those most appropriate for the existing diathesis. Need it be said that bleeding and antimony are not the means by which scorbutic or purpuric bronchitis is most effectually controlled: that, where jaundice or glycohæmia exist, those agents are inappropriate? Even in gouty and rheumatic bronchitis and in that attending Bright's disease, he antiphlogistic plan is of subsidiary importance, and only directly applicable, where the inflammatory action is of extreme violence and assumes the importance of an idiopathic disease.

606. In bronchitis of blood-origin the sputa very constantly contain some of the *materies morbi* circulating with the blood. Thus in icteric bronchitis the colouring matter of the bile often tinges the expectoration, sometimes deeply enough to be visible to the naked eye, sometimes so slightly as to require micro-chemical examination, for its discovery. Blood-discs escape with the secretion of purpuric and scorbutic bronchitis; sugar has been discovered in that of the glycohæmic or diabetic variety;* and it is infinitely probable urea is expectorated in uræmic bronchitis.

607. We must content ourselves with these general indications of the peculiarities of bronchitis dependent on blood-diseases, and with a brief description of the syphilitic variety.

SYPHILITIC BRONCHITIS.

608. That the virus of syphilis may affect the bronchi, has been made very evident by the enquiries of Drs. Graves, Stokes and Munk. It appears that a certain time after infection, febrile action and bronchial irritation occur in a variable number of

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* Francis, Lond. Med. Gaz., Feb. 1847.

cases, as preludes to cutaneous eruption, disappearing wholly or partly when this is established; and, conversely, if a syphilitic eruption suddenly disappear, spontaneously or through treatment, bronchitis may ensue.

609. Under these circumstances, the diagnosis is easy. But persons, poisoned to the secondary and tertiary degrees by syphilis, may have chronic bronchitis, as a persistent state,—they may cough, have sero-purulent and muco-purulent expectoration, nocturnal perspiration, and hectic fever, while they rapidly lose flesh and strength; and no tubercle shall exist in the lungs. Yet here is assuredly enough to create a strong suspicion of its existence, taken in conjunction with the indubitable tendency of syphilis *plus* mercury to induce the outbreak of phthisis in a person having the requisite constitutional aptitude. How are the cases to be distinguished? By the total want of accordance between the physical signs and the constitutional symptoms; the patient with syphilitic bronchitis has neither consolidation signs, nor, *à fortiori*, those of excavation. But there is a curious source of difficulty, which sometimes starts up in these cases, and renders doubt imperative: the infra-clavicular ribs and clavicle thicken from periosteitis, and produce dulness under percussion, which cannot with positiveness be distinguished from that of tubercle within the lung. Here the observer must wait for events to clear up the diagnosis.*

610. In managing this variety of bronchitis, the whole secret

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* Smedley, U. C. H. Females, vol. i. p. 143 (1846). When admitted in 1846, this woman, in addition to secondary and tertiary syphilis, and cancer of the rectum, had bronchitis and very slight dulness, with harsh respiration, under the right clavicle; but as the subjacent bones were obviously thickened, I abstained from giving any positive opinion as to the existence of consolidation of the lung. The chest-symptoms totally disappeared for the time under treatment. I have frequently seen the patient since; consolidation-signs, growing at each interval more marked, had become positive at both apices when I last (winter of 1850) saw her.

consists in having proper regard to the existing diathetic state. Ioduretted inhalations are serviceable.

BRONCHITIS SECONDARY TO CHEST DISEASES.

611. The clinical features of bronchitis thus produced will be sufficiently described with the various pulmonary and cardiac diseases originating it.

IV.—BRONCHIAL ASTHMA.

612. Bronchial asthma may depend on a *plus* or a *minus* state of the contractility of the muscular fibres of the bronchial tubes;—in the former case it is spasmodic, in the latter paralytic.

SPASMODIC BRONCHIAL ASTHMA.

613. By spasmodic asthma is clinically understood paroxysmal dyspnœa, immediately dependent on more or less extensive obstruction of the smaller bronchi, itself caused by tonic contraction of the circular fibres. The immediate cause of this contraction is, in turn, under all circumstances, perverted innervation, affecting the trunk or branches of the vagi or sympathetic nerves. And lastly this perversion of nervous influence may arise independently of, or on the other hand in direct connection with, anatomical change in the bronchi or parenchyma. Spasmodic asthma is in other words a primary or a secondary neurosis of the tubes.

614. When a primary neurosis, the paroxysms may be of direct or reflex mechanism: direct, when dependent on centric excitation in the medulla oblongata, or irritation of the vagi or sympathetic nerves; reflex, where sequential to gastric or intestinal disturbance, uterine or ovarian irritation, spinal irritability, intercostal neuralgia, hepatic congestion and hepatalgia and pure hysteria.

When a secondary neurosis, the paroxysms are excited through the reflex irritant influence of bronchitis or emphysema. The bronchitis is chronic, commonly sub-acute, and in nowise specially characterised anatomically: and as multitudes of similar cases of chronic alteration of the tubes exist unattended with asthma, there is evidently something special in the diathesis of those who suffer. In these associated cases the patient may first have been the subject of pure spasm, and eventually become bronchitic; or bronchitis may have led the way, and by degrees generated the nervous irritability. The connection of emphysema with spasmodic seizures is certainly sometimes, possibly always, dependent on intervening irritative or passive congestion of the tubes.

615. The extrinsic causes of a paroxysm in the two classes differ. In the first, where purely neurotic, any sudden impression on the nervous system will suffice. In the second, some direct irritation of the mucous surface is generally discoverable, —the inhalation of air irritant from its chemical or physical composition, from its temperature, its weight and various other qualities. But no one condition is uniformly productive of paroxysm in different persons, nor even on all occasions in the same person. Any mechanical difficulty in the pulmonary circulation, such as arises from mitral disease, or from distended stomach and bowels pushing up the diaphragm and encroaching on the chest, facilitates the action of more direct causes.

616. A paroxysm may, then, be preceded by a variety of disturbances, digestive or other, or take place independently of any one of them. Under either class of circumstances a sensation of oppression and discomfort in the chest may be the immediate precursor of the fit, or this may occur suddenly without the very slightest warning. As in all diseases where the excito-motory system plays an important part, seizures frequently take place during sleep.

617. The symptoms are then perhaps the most strikingly defined. The patient starts out of sleep with a feeling of

suffocation or constriction about the chest; the efforts at inspiration, convulsively violent, are accompanied with sinking of the epigastrium, falling in of the lower part of the sternum, and elevation of the diaphragm,—evidently from the diminished mass of air in the lungs; the expiration is prolonged and comparatively easy; both acts, but chiefly the former, are attended with wheezing. Various postures are assumed to facilitate the attempt at filling the chest,—the patient stands erect, or leans the head forwards between the hands, or places himself on his knees and elbows, or rushes to an open window, and gasps wildly for air. The pulse is small and feeble, sometimes irregular, whether there be or be not palpitation of the heart, the eyes prominent and staring, the face flushed, livid or pale, the extremities cold, the skin clammy, the look anxious and imploring. The urine, pale and abundant during the paroxysm, becomes scanty and high-coloured at its close. The fit may last from two to four hours, terminating by expectoration or not (humid and dry varieties), and leaving, on its cessation, a sensation of fatigue and prostration.

618. The physical signs are rather negatively than positively valuable. There is much laboured and jerking elevation-movement of the thorax; little or no true inspiratory expansion: retraction, on the contrary, of the anterior base may attend inspiration, while expiration, removing the abnormal retraction, may be said *pro tanto*, to expand the walls. The gradual evacuation of the supplementary, and in some measure even of the residual air, which takes place in both lungs during the paroxysm, slightly impairs the resonance under percussion.* True inspiratory sound may be almost completely deficient, sibilant or sonorous rhonchus taking its place. As Laennec pointed out, if the patient be directed to speak, without drawing breath, as long as he possibly can, so as to exhaust the chest as

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* This I repeatedly ascertained in a girl, named Harmer, a patient both of the Consumption and U. C. Hospitals; but, on the whole, this sign is rarely to be established.

completely as possible, the next two or three quiet inspirations produce well-marked and very natural respiration-sound: obviously the tidal air advances deeply into the tubes, and under these special circumstances may be supposed even to reach the vesicles; at all events a fall back of the residual air occurs. This can only depend on temporary relaxation of spasm: but why the process described should effect such relaxation, is not so clear.

619. Where the disease is purely neurotic, the patient during the intervals between the seizures, may enjoy very perfect health, as far as the lungs and breathing are concerned. Where, on the other hand, chronic bronchitis, or emphysema exist, the symptoms of these states are more or less obscurely, their physical signs very plainly, to be discovered. Subjectively, however, even in these cases the intervals may be passed by the patient in comfort and unconsciousness of ailment; it is not uncommon to hear habitual asthmatics boast of their perfect breath, at a time when wheezing is audible at a distance, and all the physical signs of emphysema and chronic bronchitis exist in perfection. Habit, the great physiological modifier, gradually reconciles the system to a minimum supply of oxygen.*

Repetition of asthmatic fits may lead to dilatation of the right heart, and insufficiency, without structural change, of the tricuspid valve; but this is very rare, unless there be emphysema, as a permanent malady.

620. The treatment of spasmodic asthma embraces that of the fits, and of the constitutional tendency to their recurrence.

621. In the fit, our effort must be to relax spasm. In a first attack, if there be great congestion, bleeding may be advisable; but in the case of habitual seizures, it is altogether inadmissible. Nauseating expectorants, ipecacuanaha, lobelia, squill, or

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* Hugot Arnott, it is said, was one day, while panting with asthma, looking out of his window, and was almost deafened by the noise of a bawling fellow who was selling oysters; "Tho extravagant rascal!" said Hugot; "he has wasted in two seconds as much breath as would have served me for a month."

colchicum (if the patient be gouty), or an emetic of sulphate of zinc, sometimes cut short a fit very obviously; various anti-spasmodics, sulphuric æther, asafœtida, musk, and very strong coffee mitigate the intensity of the paroxysm. Of narcotics, stramonium, cannabis indica, belladonna, and opium, are the most effectual. Inhalation of the fumes of burning paper, saturated with nitre, sometimes shortens a paroxysm, by nducing copious expectoration; true, it oftener fails; but I have known old sufferers from asthma confide implicitly in it, to the exclusion of other remedies. The electro-galvanic current, directed from the nape of the neck to the epigastrium; the cold-water-dash over the shoulders; draughts of cold water, while the feet are immersed in a mustard foot-bath,—are all agencies that occasionally relieve.

The effects of chloroform inhalation, commenced after the nvasion of the fit, pushed to narcotism, are variable. I have seen three kinds of result: total relaxation of the spasm during the continuance of insensibility, with immediate return of dyspnœa on restoration of consciousness,—gradual return of the difficult breathing, after consciousness is restored,—and suspension, or at least mitigation, of the paroxysm for the time being. The last effect is the rarest of the three; but, on the other hand, the temporary relief afforded by chloroform is sometimes more complete and more rapid than that effected by any other agent. Small quantities of chloroform, inhaled when the sensations precursory to a fit are felt, sometimes effectually ward this off; and there is reason to believe that, in some cases at least, the repeated use of chloroform after this plan, may eventually break the paroxysmal habit altogether:* this effect can, however, only be hoped for, where the disease is a pure neurosis.

A coming fit may, also, be averted by a cigarette of stramonium and belladonna, by strong coffee, by mental excitement or amusement, or by heating the body generally to as high a degree as can be borne. Sedatives and excitants of the peripheral nerves seem to have the same influence on those of the lungs.

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* J. R. Reynolds, M.D., in "Lancet," Oct. 29, 1853.

622. The tendency to paroxysms may sometimes be removed by counter-irritants to the dorsal spine, or to the nucha, by strychnia in very minute doses, by electro-galvanism steadily repeated in the course of the vagi and phrenic nerves, by great attention to the state of the alimentary canal, caution in eating both as to quantity and quality, and the use of certain metallic tonics, especially the chloride or iodide of arsenic, the nitrate or oxide of silver, and the valerianate or sulphate of zinc. If there be chronic bronchitis, the measures, advisable for that disease, must of course be enforced.

Change of air is most important; but the kind of change that shall prove beneficial can only be learned by experience. Some sufferers lose their paroxysms south of the olive line, others are easiest in a cold atmosphere; moisture, the bane of some, greatly mitigates the disease in others; any notable fall in the barometer induces a paroxysm in one, wards it off in another. The air of towns suits some, that of the country others; the relatively clear suburban air of London is infinitely more noxious to some asthmatic persons than the foul atmosphere of the worst-cleansed and most densely-peopled localities of the metropolis; * occasionally an individual will be found who is tortured with asthma in one room of a house, free from it in

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* A man, one of the greatest sufferers from asthma I ever saw, lived in the neighbourhood of Chalk Farm, the pure air of Hampstead blowing across his house. I tried, I believe, almost every known remedy, in vain, for his relief. He was accidentally detained one night in the foul region of the Seven Dials; feeling persuaded he could not possibly survive till morning, so great was his dread of the close atmosphere. He not only lived through the night, however, but enjoyed the first uninterrupted sleep he had known for months. He took the hint; removed to the Seven Dials for *the benefit of the air;* and when I last saw him, some six months after the removal, continued, though still a wheezer, perfectly free from serious dyspnœa.

others,—and this without any distinct explanation being found in the aspect, the drainage, or any other known condition.

PARALYTIC BRONCHIAL ASTHMA.

622*. Laennec ascribed the peculiar air-distension of the lungs found in persons asphyxiated by the mephitic gases of cess-pools, to paralysis of the vagi nerves; Mr. Swan noticed similar distension in animals whose eighth pair had been divided in the neck. In both cases the contractile force of the bronchial muscles, concerned in expiration, is more or less completely annulled.

623. If then, as we have seen, there be motive to believe that nervous asthma commonly depends on spasmodic action of the bronchial muscular apparatus, here are speculative reasons for presuming that paralysis of the apparatus may cause a variety of the affection. Clinically, too, we meet with examples of asthma in which the comparative facility of inspiration, and difficulty of expiration, suggest of themselves the probability of a minus rather than a plus state of power in bronchial contractibility. Possibly such cases are those benefitted by strychnia and galvanism [555].

HÆMIC ASTHMA.

624. Difficult or distressed breathing, produced by morbid conditions of the blood, is far from rare. Some of its varieties deserve brief mention here, were it only for the purpose of guarding the student against errors of diagnosis. These morbid states, probably, interfere with ready oxygenation directly or indirectly; and the breathing is instinctively increased in frequency to make up for the deficient amount of oxygen supplied to the system by each separate inspiration.

625. The chief species of hæmic dyspnœa are the anæmic, the uræmic,* the icteric, the pyohæmic, the gouty, the saccharine or glycohæmic, and the choleraic.

626. In all these species the dyspnœa has much the

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* Association Med. Journal, Nov., 1853, p. 1010.

character of the breathlessness following over-exertion in health; the respiration is, absolutely speaking, not frequent in the ratio of its apparent labour; it is not seriously out of proportion with the pulse; and the subjective sensation of want of breath is comparatively little felt. There are no physical signs, cardiac or pulmonary, to explain the morbid state of the breathing; and the specific evidences of the several diseases named are readily discoverable.

ORGANIC CHANGES OF THE BRONCHI.

NARROWING OR OBLITERATION OF THE BRONCHI.

627. Narrowing and obliteration of the bronchi, a common phenomenon in tubes of very small calibre, becomes rarer and rarer directly as their size; still, obliteration, even of the main trunk, has occasionally been witnessed. The obstruction may depend on *intrinsic* causes,—such as thickening of the mucous membrane, stagnating secretions, especially of the plastic kind, (under both these circumstances the condition is of inflammatory origin), or accumulation of tubercle or cancer in their interior: or obstruction may be produced by *extrinsic* pressure,—for instance, that of adjacent emphysema, adjacent tuberculous deposit, plastic contractile exudation-matter, infiltrated cancer or chronic solid pleural formations; enlarged bronchial glands, aortic aneurisms and mediastinal tumors have sometimes effected the closure of a main trunk by pressure.

628. If obstruction of a large bronchus, by its own secretions, occur suddenly, or even with notable celerity, dyspnœa, proportional to the size of the implicated tube, follows; and as this has chiefly occurred in cases of bronchitis, where the efficient breathing surface had already been seriously diminished, risk of fatal asphyxia is incurred, unless the patient retains strength sufficient to enable him to expectorate the stagnating matters: the relief experienced after such expectoration is almost assimilable to that following tracheotomy for obstructed larynx.

But if the obstruction be on a small scale, or if a large tube suffer only from slow, gradually increasing obstruction, there will be no positive subjective or objective symptoms to indicate the occurrence. Such dyspnœa, as is really dependent on obstruction of the kind, appears explicable by other conditions; and neither cough nor pain in the chest, to an amount unusual to the patient, are induced.

629. The physical signs may or may not be satisfactory. Obstruction of the main tube produces collapse of the entire lung, as is well shown in one of Sir R. Carswell's published drawings; * obstruction of minor tubes, as insisted on by Dr. Stokes, produces local and limited collapse,—the surface of the lung in the affected parts appearing sunken in below the plane of the surrounding pleura. Now, in both these varieties percussion-dulness, proportional to the superficial extent and depth of the collapse, would constantly exist, were it not that, in the local variety, emphysema tends to spring up on the confines of the collapsed spots, and give rise to its own special clear resonance. The respiratory murmurs are either actually suppressed or weakened considerably, and harsh; and mingled with sonoro-sibilant rhonchi.

630. The main interest, in the present state of knowledge, of obstruction of a large bronchus, arises from the light it may throw on the diagnosis of thoracic aneurism and tumor.

631. The treatment of the affection is altogether that of the disease of which it is a sequence or effect.

DILATATION OF THE BRONCHI.

632. Dilatation of the bronchi has already been considered in its association with chronic bronchitis; in its secondary relationship to acute and chronic pneumonia, phthisis, and pleurisy, it will be referred to in the description of those diseases.

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* Fasc. Atrophy, pl. iv., fig. 3. The lungs are those of a monkey,—the cause of obstruction, extensively tuberculised bronchial glands. Atrophy from inaction would doubtless ensue after a time.

TUBERCLE OF THE BRONCHI.

633. Tuberculous disease of the bronchi is limited to the finer tubes. The morbid matter, as Sir R. Carswell was the first to show, occasionally forms on the free surface of their mucous membrane: but he was in error in imagining this to be its essential seat; and bronchial tuberculisation, as clinically distinguishable from that of the parenchyma, is unknown.

CANCER OF THE BRONCHI.

634. Primary cancer of the bronchi, or cancer affecting these tubes independently of similar disease in the lungs or mediastina, is not observed. But the following secondary forms of the disease are met with.

1. In certain cases of cancerous infiltration and ulceration of the pulmonary tissue, those morbid processes spread to, and cause the destruction of, the bronchial tubes involved. 2. Perforation of a bronchus by a portion of actively growing tumor situated about the root of the lung, has occurred in rare instances; the perforating cancer vegetates freely in the interior of the tube into which it has made its way. 3. More rarely still, a detached tumor forms in connexion with the mucous membrane of a bronchus, the lung being similarly diseased. 4. Cancerous substance may be found *in transitu* outwards from a softening pulmonary mass.

All these forms of disease derive their practical importance from their connexion with cancer of the pulmonary substance, and therefore require no further consideration in the present place.

SECTION III.—DISEASES OF THE BRONCHIAL GLANDS.

TUBERCULISATION, OR BRONCHIAL PHTHISIS.

635. Tuberculisation of the bronchial glands, an affection practically speaking, almost confined to childhood, occurs

commonly in the chronic, with infinite rarity in the acute form.

CHRONIC TUBERCULISATION.

636. All varieties of tuberculous deposit occur in these glands; the gray granulation is on the whole rare, the yellowish mass, infiltrating the tissue, the most common form. Degeneration commences indifferently at all parts of the glands; the changes undergone by tubercle are the same here as elsewhere: partial cretaceous transformation is not very uncommon. Evacuation of softened tuberculous matter may take place into the pulmonary substance, into the trachea, into a bronchus, or into the œsophagus: a permanent fistula is sometimes established. Cretaceous matter may be expectorated from the interior of a bronchial gland.*

637. Tuberculised glands vary in size from the natural bulk to that of a walnut. A few only, or several, may be affected: in the latter case, those adjoining the bifurcation of the trachea may form an irregularly rounded lobulated mass, two, and even more, inches in diameter. The glands adjoining the base of the heart, and those of the posterior mediastinum, undergo similar, though, generally speaking, less marked enlargement. Those seated in the substance of the lung rarely acquire large dimensions.

638. Tuberculised bronchial glands may produce, according to their bulk and the direction of their growth, more or less marked compression, and eventual perforation of various adjacent structures. A general description of these compressions and perforations will be found in the section on Diseases of the Mediastina.

639. An excavated bronchial gland, sunk deeply into the lung-substance, simulates, and has doubtless often passed for, a true pulmonary cavity,—a fact first pointed out by MM. Rilliet and Barthez.

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* Carswell's Illustrations, Tubercle, pl. iv., fig. 4.

640. Tuberculisation of the bronchial glands is either associated, or not, with pulmonary tubercle. In the former, by far the more common case, the bronchial disease may be the chief evidence of the diathesis, or be of altogether secondary importance. In point of time the bronchial glands may be affected prior to, or subsequent to, the lungs, whether the clinical importance of the glandular affection be trivial or most serious. The cervical, inguinal, and mesenteric glands are in some cases tuberculised.

641. The symptoms and signs of bronchial phthisis are those of bronchitis, mediastinal tumor and the tuberculous diathesis, variously and capriciously associated: the symptoms are, locally, irritative and mechanical, and, constitutionally, hectic.

642. The local symptoms of mechanical origin are substantially the same as those of mediastinal tumor in general (vide Section VI.), and depend on pressure, traction, or perforation of the various parts there enumerated. Among the most important of these symptoms are swollen cervical veins, lividity and œdema of the face, neck, and upper part of the chest,—limited to one, or affecting both sides, according as either vena innominata or the superior vena cava is compressed: I have never seen distinct œdema of the arm. Discharge of blood by the mouth or anus is rarely observed,—hæmoptysis especially very seldom seen in children under the tenth or twelfth year: but the escape of blood into and from the pulmonary texture through pressure is, we have every reason to believe, of tolerably common occurrence; it is swallowed as it reaches the mouth. Spasmodic dysphagia from pressure on the œsophagus has occasionally been witnessed. Heaviness and stupor, from obstruction to the return of blood from the brain, are common; and pressure either on the trachea, bronchi, the eighth pair, or recurrent nerves, causes singular modifications of the voice, cough, and physical signs, to be presently described.

643. If the mass of tuberculised glands be large, and press sufficiently on the root of the trachea, or on both main bronchi,

o impede the entry of air, the lower lobes gradually collapse, he chest falls in latero-inferiorly on both sides, and the sternum nd costal cartilages come forwards: the infra-mammary and infra-axillary regions expand imperfectly, the infra-clavicular in excess. If the main bronchus on one side only be obstructed, flattening of the surface will be limited to that side.

The vocal fremitus is often in excess in the inter-scapular region.

The essential physical sign, however, is percussion-dulness between the scapulæ; taken simply, by far the most significant of the series. I have known the resonance greatly impaired in front from the base of the heart to the sternal notch,*— but this is rare. The percussion note is highly tubular between the scapulæ in some cases. Where a main bronchus has been sufficiently pressed upon to induce general collapse of its communicating lung,† the side generally is deficient in tone, especially under broad percussion; but the dulness is not so marked as might *à priori* be expected.

The auscultation-signs vary widely under different circumstances. If there be sustained pressure on the trachea or large bronchi, sonoro-sibilant or liquid bronchial rhonchus, and weak almost suppressed respiration of intermittent type, and of unnatural but variable quality, are heard. At the same time laryngo-tracheal stridor and rattle may be audible at a distance. If there be no pressure, and the glands adhere closely both to the chest wall and the bronchi, high-pitched, hollow, bronchial respiration, large-sized, ringing, liquid rhonchi, and powerful quasi-pectoriloquous bronchophony are audible with varying perfection and steadiness. Lastly, if there be no pressure by the glands, and if no artificial medium of conduction between the tubes and the surface be formed by them, the auscultatory signs will be null, or those of simple bronchitis.

644. The child affected with bronchial phthisis, lies, sits or leans in all varieties of postures: orthopnœa is the rule in

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* Case of S. T., ætatis 6, seen with Mr. C. Crosswell.

† Carswell's Elementary Illustrations, Atrophy, pl. iv., fig. 3.

paroxysms of difficult breathing. Often there is no evidence of thoracic pain or tenderness; if there be marked suffering, this depends on some superadded, generally pleuritic, irritation. Cough, an almost invariable symptom, may be totally devoid of significant character; or, hoarse, ringing, dry, occurring in fits resembling those of hooping-cough, but not accompanied with actual hoop, nor followed by ropy expectoration and sickness, it may give clear indication of dependence on irritative pressure of the vagus or recurrent nerves. So, too, the voice and cry may retain their natural characters, or become hoarse, feeble, or even temporarily null. If expectoration occur, the sputa are simply catarrhal, muco-epithelial or purulent; in the adult, tuberculous matter in all stages may be expectorated, if a diseased gland open into the bronchial tubes. The respiration, varying between twenty and eighty per minute, may be steadily and uniformly shallow and laborious; or paroxysms of dyspnœa, accompanied with lividity of the face, anxious countenance, cold clammy perspiration, and all the phenomena of partial asphyxia, may supervene from time to time. In one case of this kind, in a child aged three years, the characters of the seizure were very much those of laryngismus stridulus. I have never seen actual convulsions.

645. The general symptoms, insignificant perhaps at the outset, eventually become those of pulmonary phthisis: hectic fever, with night perspirations, loss of strength, colliquative diarrhœa, and excessive emaciation,—symptoms commonly referrible in part, however, to advancing general or pulmonary tuberculisation.

646. The ordinary mode of death by gradual asthenia, is sometimes varied by accidental occurrences, such as tremendous hæmorrhage from perforation of the pulmonary artery, pneumothorax, or meningeal hæmorrhage.* Pneumo-thorax is not necessarily immediately fatal.

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* I give this cause of death, of which I have not met with an example under the circumstances, on the authority of MM. Rilliet and Barthez.

647. The direct diagnosis of bronchial phthisis can only be made through the association of several of the local and mechanical symptoms with those of constitutional character, and in the majority of cases, the disease has far advanced, before such kind of diagnosis becomes possible; the method, more commonly guiding to its detection, is indirect. Thus, if a scrofulous-looking child become feverish, and cough more or less steadily, and if the physical signs cannot be referred to bronchitis, or pulmonary phthisis, while there is no evidence of mesenteric or encephalic tuberculisation, a strong suspicion may be entertained that the bronchial glands are the parts affected. The suspicion will be powerfully strengthened, if even trifling percussion-dulness can be detected between the scapulæ, and converted into certainty, if the cough become paroxysmal, and signs of venous pressure supervene. The auscultation-signs vary so much in different individuals, and in the same case even, that they are entitled to little confidence; still their very irregularity excludes other diseases.

648. The treatment, being essentially that of pulmonary phthisis, need not here be dwelt upon. I would simply observe that from the free internal use of cod-liver oil, and of ioduretted applications between the shoulders, complete restoration to health, combined with physical evidence of diminished bulk of the bronchial glands, may sometimes be accomplished.*

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* One of the most striking examples I have met with of such recovery occurred many years ago among the out-patients of University College Hospital. Two children, aged about five and seven years, who had just lost their mother from phthisis, were brought to the hospital, one in arms, by their father. Both were in the last stage of emaciation, had mesenteric disease, all the rational symptoms of phthisis, and marked inter-scapular dulness under percussion; they rapidly recovered flesh and strength, and when they ceased to attend, had not only lost all their chest-symptoms, but had become normally resonant, or very nearly so, between the shoulders.

ACUTE TUBERCULISATION.

649. Acute bronchial phthisis is excessively rare. MM. Rilliet and Barthez have, however, recorded one most unquestionable case, in which death took place in six weeks from the first appearance of illness. Paroxysmal cough, occurring five or six times daily was the first symptom noticed; the pulse and respiration were excessively frequent; progressive emaciation and copious perspirations proved the deepness of the constitutional suffering. Not a particle of tubercle was found in the lungs; and a single crude miliary tubercle in the brain, without surrounding softness, was the sole specimen of that product in the body, in addition to those in the bronchial glands.

SECTION IV.—DISEASES OF THE PLEURA.

I.—HÆMOTHORAX.

650. Pleural hæmorrhage, or hæmothorax, is in the majority of cases traumatic, and results from wounds or violent contusions of the walls of the chest, and fractures of the ribs. When non-traumatic, it is always a secondary state; for of pure idiopathic, passive or active, hæmorrhage into the pleural cavity, I have never heard of a positive example. The fluid of pleurisy contains blood in considerable quantities in some cases; but no difference is thereby impressed on the physical signs, or (notwithstanding the opinion of Laennec) perceptibly on the progress of the disease. In various blood-diseases with hæmorrhagic tendency, as scorbutus and purpura, if hydrothorax occur, the serous pleural effusion is more or less stained with blood. Carcinoma of the lung bursting into the pleura, or carcinoma of the pleura itself undergoing superficial ruptures,* or apoplexy of the lung, making its way through the pulmonary pleura, or rupture of an aneurism, may all produce hæmothorax.

651. Whether traumatic, or an effect of internal disease, the

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* Dewing, U. C. H., Males, vol. v., p. 19.

sole symptom clearly assignable to hæmothorax is laboured breathing,—the mechanical result of pressure of the lung. Various coexisting symptoms are the effects of the injury or internal disease, on which the hæmorrhage itself depends.

652. The physical signs of traumatic hæmothorax are dulness under percussion, enfeebled or annulled vocal fremitus, weak or suppressed respiration, absence of rhonchus, the vocal resonance being in a variable state, null, bronchophonic, or occasionally ægophonic,—signs coming on suddenly without inflammation and under circumstances of injury to the chest.

In cases where extensive accumulation of blood occurs in the pleura from any of the internal diseases named, similar physical signs will be observed,—but they may be rendered obscure and uncertain by the primary disease. Hæmothorax of such origin may be abundant enough to drive the heart greatly out of its place. Thus, in a case of aneurism of the arch of the aorta adhering to the left lung, in which an effusion of about two quarts of blood into the pleura of that side produced sudden death, the heart was found lying to the right of the middle line.*

653. The absence of friction-sound will at the outset distinguish hæmothorax from pleuritic effusion. Hydrothorax is characterised by affecting both pleural sacs almost simultaneously, and hence, whatever be the amount of percussion-dulness and fluid accumulation, by its not seriously displacing the heart sidewards. The direct physical signs are the same in the two varieties of effusion: and the nature of the pre-existing internal disease will afford the main safeguard against error.

654. The treatment of hæmothorax only requires special consideration in its traumatic variety. Paracentesis is strongly recommended by surgeons of extensive military experience, at two conjunctures. First, at the outset of the hæmorrhage, if the quantity of blood effused be sufficient to threaten asphyxia

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* Bell, U. C. H., Males, vol. vii., p. 169.

by pressure on the mediastinum and opposite lung. But as the pressure of the extravasated blood against the bleeding vessels acts probably as a sort of mechanical obstacle to further hæmorrhage, a small quantity only of the fluid must be allowed to escape, the operation being repeated when urgent symptoms again set in. Such was Larrey's practice,—and it has been successful.* Secondly, at the end of ten or twelve days, if in spite of venesection and antiphlogistic treatment generally, signs of pleuritis, followed by effusion, occur, the chest ought to be punctured. The results are certainly more favourable in traumatic, than in idiopathic cases.

II.—PLEURISY.

655. Inflammation of the pleura, pleuritis or pleurisy, runs an acute or a chronic course,—in the latter case the disease may be chronic from the outset, or supervene on the acute malady.

656. Acute pleurisy is, anatomically, signified by subserous vascular injection, of the arborescent and capilliform varieties; tufts of capillary vessels elevate the membrane from spot to spot, and shoot into its substance. The membrane loses its natural moisture and smoothness, its elasticity, transparence and consistence, sometimes even its natural thickness from separation of epithelium. Under special circumstances it is locally destroyed by gangrene. The sub-pleural cellular tissue is injected, softened, œdematous, or, in rare instances, infiltrated with pus. The pleural sac contains solid, liquid, or gaseous new products. The solid material consists of exudation-matter, which vascularises rapidly (sometimes in thirty-six hours), and which may be completely absorbed, or gradually undergo conversion into induration-matter, or into pseudo-cellular tissue. Secondarily, there may occur in its substance calcification or pseudo-ossification; and various species of the genus Deposits, as

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* M. Roux successfully removed three pounds of blood, extravasated in a case of fractured ribs; quoted by Sédillot, de l'Empyème, p. 107.

pus, tubercle and melanic matter, may form within it. The fluid contents, so-called serous effusion, consist of water holding in suspension exudation-corpuscles, pus-cells, epithelium, and frequently blood-discs, discoverable with the microscope; any one of these products may exist in sufficient quantity to be visible with the naked eye,—flakes of albumino-fibrinous matter and pus in all proportions. The quantity of albumen in solution varies from 31 to 77 per cent.; fibrine commonly exists in this same state also,—the contents of the sac, perfectly fluid when first seen, subsequently gelatinising from fibrine-coagulation.* The fluid may be solely purulent; but this is very rare in simple pleurisy. Unless from the accidental occurrence of sloughing, or special alteration of the fluids, gas does not form within the inflamed pleura.

657. In simple idiopathic pleurisy one side only suffers: in some diathetic affections, as tuberculous disease, Bright's disease, and scorbutus, both pleuræ may be attacked simultaneously. The surface of either sac may be generally, or only partially, inflamed: in the latter case, the pleurisy is called phrenic, pulmonary, costal, interlobar or mediastinal, according to its precise seat. In some cases corresponding, but limited portions of the costal and pulmonary pleuræ are affected, and thus arises the circumscribed or locular variety of the disease: two or more such circumscribed pleural collections may form, whence the terms bilocular and multilocular.

658. The various appearances, now referred to, are found to occur, as a rule, in fixed sequence; hence have been established certain anatomical stages of pleurisy,—stages the more important, because they correspond with precision to striking

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* This I have seen take place in pleuritic fluid in from half an hour to an hour after its removal from the chest. Sometimes a second coagulation occurs much later, from twenty-four to thirty-six hours, from the presence of pseudo-fibrine, or "fibrine of retarded coagulation," as it has been called by Virchow. In a case of this kind Schlossberger suggests the retardation may have been due to some urea existing in the fluid. "Edinb. Jour.," Oct. 1849.

changes in the physical signs, and even symptoms, of the disease. These stages are called the dry, the plastic, the effusive, and the absorptive.

659. (*a*) In the dry stage, vascularity and loss of natural humid polish of the pleural surfaces are the only morbid changes. (*b*) In the plastic stage, exudation-matter forms in adherent patches or layers, or loose flocculent masses; sometimes on a sufficiently extensive scale to coat the entire lung. (*c*) In the effusive stage, serosity or purulent fluid, with the characters already described, accumulates, commencing at the base of the chest.* From the moment such accumulation sets in, the lung begins to suffer pressure,—pressure which may eventually condense the organ into a small elongated flattened mass, lying against the spine. The lung is then either partially or generally airless (even its persistent air [82] being forced from the cells), tough and leathery to the feel, of slatey gray hue on section, homogeneous looking, and non-granular, and of such density as to sink instantly in water. The forcible contraction of a coating of plastic exudation sometimes aids the fluid pressure in thus emptying the lung absolutely of its air. (*d*) In the absorptive stage, the morbid products are removed by absorption,—the lung at the same time gradually recovering its expansibility, or remaining more or less condensed. The latter condition will occur, when the pressure of the lung has been long maintained; and especially when the plastic exudation embracing the organ has passed into the condition of induration-matter; the expansion-force of inspiration is unable to overcome the solid resistance offered by a lamella of the toughness, firmness, and slow

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* M. Woillez's division of the effusive stage into two sub-stages, the *laminar* and *gravitating*, though founded so strictly on physical principles, that there is great difficulty in contesting its justness, may be ignored in a practical work, because clinically it cannot be substantiated; at least, I have never succeeded in finding the signs of a thin sheet of fluid equally spread over the entire lung, from apex to base. The time during which the suction of the lung is a force more powerful than gravitation of the fluid must be exceedingly brief.

iolecular contractility possessed by that species of morbid roduct.

The stages now passed in review are those of the *acute* disease.

660. When the lung remains permanently more or less lattened and airless from the causes just explained, the thoracic valls undergo proportional retraction or depression, and the iffected side ceases almost totally to take part in the process of espiration. Still as inefficiency of one side may be made up for y extra-activity of the other, so long as the patient has no local uffering, and his general health maintains its past average, this ondition cannot *per se* be clinically set down as a form of hronic pleurisy. However a pleural sac in this physical and natomical state is exceedingly prone to fresh irritation,—the erous membrane itself, and even the vascularised exudation-matter, inflame subacutely, or even suppurate from time to time; or tubercle * is deposited within the diseased sac: here is one form of *chronic* pleurisy.

Again, sometimes, absorption fails to take place in the ordinary way in the acute disease; or secretion continues relatively so active, that the pleural sac remains more or less distended with fluid: this unfavourable course is more particularly observed in diathetic states,—such as those of phthisis, Bright's disease, and carcinoma,—the lung, reduced to its minimum bulk by pressure, loses substance by a process of atrophy. The fluid is under such circumstances generally purulent; † in protracted cases the ribs, sternum, or vertebræ become carious, or the ribs may become the seat of interstitial hypertrophy and sub-periosteal osteophytic growth; ‡ the costal pleura may undergo perforation, and sub-cutaneous abscess with fistula, eventually opening externally, and daily discharging for months or years, a variable quantity of pus, may form. Sloughing of the pleural induration-matter under the latter conditions occurs from time to time; or this change may affect the pyogenic membrane lining the sac of induration-matter: melanic pigment is sometimes formed in large quantity in the sac.*

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* Univ. Coll. Museum, Models, No. 5000.

† In 162 cases of pleuritic effusion, collected from different sources by Krause (das Empyem, p. 33), the fluid was purulent in 101, yellowish serous in 41, and reddish serous in 20.

‡ U. C. Museum, No. 4067.

661. This brief outline of the anatomical characters of the various stages of the disease will render the course of the physical signs intelligible.

662. *Physical Signs. Dry Stage.*—The motions of expansion and of elevation, from the consensual avoidance of pain, are diminished on the affected side visibly, palpably, and by semicircular and antero-posterior measurement; their rhythm is slightly jerking. The percussion-sound is not perceptibly duller than natural; I have never known its pitch raised. The respiration is weak, but superficial, and jerking in rhythm. Grazing friction-sound may often be caught, especially about the infra-mammary and infra-axillary regions: in rare cases loud, rubbing friction-sound may be audible over the entire side, without a particle of exudation-matter having been thrown out.†

663. *Plastic Exudation Stage.*—The state of chest-movement continues as before; rubbing vibration may sometimes be felt with the hand, but is very rarely perceptible to the patient: the vocal fremitus continues natural. The clearness of percussion-sound is somewhat diminished; if it be at all notably impaired and the sensation of resistance be even slightly increased, the

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* Wicks, U. C. H., Males, vol. i., p. 61.

† I base this statement on the following case:—A man, dying of cardiac disease, presented on August 4, "all over the front and side of the left chest up to the first rib, grating and creaking friction-sound, accompanied with marked friction-fremitus; the patient is well conscious of this rubbing, but feels not the slightest pain." On this man's death, which occurred sixteen days later, fluid, which was known to have formed, appeared in the pleura; but the membrane was "entirely free from lymph, except over a spot about the size of half-a-crown at the angle of the seventh rib; this lymph is not rough, and does not look recent. The entire pleural surface is highly vascular, especially along the ribs and at their angles; the vascularity gives roughness to the surface." J. Wilkinson, U. C. H., Males, vol. ix., pp. 284, 306.

plastic exudation is abundant: deep inspiration will restore the sound to its natural character. The respiration continues of weak, jerking type; and is accompanied with friction-sound of the rubbing or even grating varieties: pleural friction of cardiac rhythm may also by possibility be detected. No influence is exercised on the resonance of the voice: if pseudo-ægophony exist, it depends on some condition extraneous to the pleura.

663* *Effusive stage.*—(Without dilatation of the side.) The jerking rhythm of the movements visible in the past stages now gradually disappears; and both the expansion and the elevation classes of movement (the latter are less affected than the former) cease to be visible at the lower part of the side; the infra-mammary and infra-axillary regions become more or less bulged. The intercostal spaces, especially in the infra-lateral regions, may be less hollow, and sink in less with inspiration, than those of the healthy side. Vocal vibration, as a rule, is absolutely abolished, wherever fluid has accumulated to any amount: above the fluid, it continues perceptible: so, too, friction-fremitus, if it existed before, disappears wherever the fluid prevents the collision of the layers of exudation-matter; but may remain in full energy above the level of the effusion, or continue in front, while annulled in the back. Mensuration proves the existence of deficient expansion and retraction; but the difference of respiratory play on the affected and non-affected sides is not so great at this period of pleurisy as in more chronic conditions,—pain still interferes with the general chest-action, and the non-affected lung has not yet acquired the habit of expanding to its full limits. Meanwhile dulness under percussion becomes very manifest, with increased parietal resistance, inferiorly; the limits of the dull and clear sounding parts are distinguished by a tolerably well-defined line,—and the area of the dulness, and its line of demarcation, may be changed by altering the patient's posture,* but not by the act of respiration. In rare instances the inferior lobe of the lung loses bulk so considerably by pressure, that a fall may occur in the upper limits of dulness, in consequence of the fluid gravitating downwards to fill the otherwise vacant space; hence the area of superficial dulness may by possibility decrease, while the fluid actually increases. Tubular or amphoric resonance is found commonly at the upper front of the affected side [143], and sometimes posteriorly. Auscultation shows that the respiratory murmurs, suppressed where the effusion is most abundant, —weak, and deep-seated, where less abundant, acquire, above the level of the effusion, exaggerated force, and more or less of harsh, bronchial, or even blowing quality. Friction-sound may still be sometimes discovered above the confines of the fluid; and, when deficient in the back in the sitting posture, may still be found there if the patient be made to lie on the face for a while.* This is the most favourable period of the disease for the detection of ægophony, as already [255] fully explained: but the vocal resonance may be strongly or weakly bronchophonic, or may be absolutely null; the cases in which ægophony is actually caught are certainly in the minority,—a fact for which an explanation has already been tendered. The heart's sounds are heard with greater intensity through a given thickness of fluid, and of lung solidified by condensation, than through an equal thickness of healthy parenchyma; hence, in right pleuritic effusion, they are more clearly audible than in the natural state, in the right axillary region. Besides, the action of the heart may produce a sort of succussion or fluctuation-movement in the fluid, which if that organ be hypertrophous, is very perceptible at the opposite surface of the chest.

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* This change in position of the fluid may be ascertained by percussion, even where the quantity is small; thus, where it reaches in the sitting posture

664. *Stage of Effusion with Dilatation and Thoracic Displacements.*—The fluid having accumulated sufficiently to fill all available space in the pleural sac, increased in capacity to a

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only as high as the inferior angle of the scapula; e. g., Humbert, U. C. H., Males, vol. ix., p. 14.

* J. Harrison, U. C. H., Females, vol. ix., p. 312.

great amount by compression of the lung, begins to push the walls of that sac before it in all directions. The costal parietes, the mediastina, the diaphragm, and the triangular apex of the pleural sac above the clavicle yield before the encroaching liquid,—hence various important modifications in the physical signs. The affected side, either expanded generally, or bulged below to a high degree,—motionless inferiorly, unless a powerful inspiratory effort be made, when a slow, dragging, upward movement takes place, posterior in time to the expansion of the other side,—sometimes exhibits a notable change in the state of its intercostal spaces; these may be widened, flattened, or even convex outwards, and in the latter state may exhibit, besides, visible fluctuation. On the other hand considerable bulging of the walls may exist inferiorly in acute effusion, without any notable distension, or deficiency of inspiratory sinking in, of the interspaces; the latter may even be more obvious than on the healthy side: * nay more, in chronic effusion with considerable general expansion of the side, a fair amount of normal intercostal breathing action may be maintained,† [37*]. The thoracic surface is felt to be unnaturally smooth; all vocal vibration has absolutely disappeared; and peripheric, more rarely simple, fluctuation may be detected by the fingers in the distended intercostal spaces. The semi-circular, antero-posterior, and vertical measurements of the side, and the distance between the nipple and middle line, increase; while the respiratory play falls notably,—in fact may be null.‡ The dull percussion-sound

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[*] J. Harrison, U. C. H., Females, vol. ix., p. 312.

[†] Crowhurst, U. C. H., Males, vol. ix.

[‡] The respiratory play may be otherwise singularly perverted. Thus, in a case of left effusion (H. Morris, U. C. H., Males, vol. vi., p. 33, January 1, 1851), the seventh and eighth ribs and interspaces below the left nipple sank in during forced inspiration, both visibly and palpably, while the abdomen rose considerably; yet semicircular mensuration indicated at the same spot a respiratory play on the affected side of three-eighths of an inch (Insp. 17¼; Exp. 16⅞ inches). This is a striking discordance between antero-posterior and general expansion movement [74].

of the previous period now extends downwards, upwards as far as, and even by possibility above, the clavicle, and passing the middle line in front, encroaches at the upper part of the mediastinum on the opposite side of the chest. The displaced heart also carries its own proper dulness into the new position the fluid may have driven it. All sense of elasticity in the parietes has disappeared; and the outline of the dull sound is scarcely to be altered by changing the position of the patient.* Besides, in the great majority, at least, of cases the upper part of the chest, especially about the clavicle, first and second ribs, and adjoining part of the sternum, even to the other side of that bone, furnishes tubular or amphoric resonance. I have observed the amphoric variety in cases of latent effusion, where the patient has been walking about, and unconscious of local ailment of any kind. The respiratory sounds, totally suppressed, except close to the spine and at the apex of the lung, possess in those regions a harsh, bronchial, or even slightly blowing quality—a quality explicable by the condensed state of the lung. In many cases, however, diffused blowing respiration is pretty extensively heard over the diseased side.† Friction-sound, in rare instances, still continues audible in some limited spot: the rule is, that it totally disappears. The continuance either of ægophony or any other form of vocal resonance is unusual; but strong bronchophony about the inferior angle of the scapula may coexist with abundant effusion, and annulled vocal fremitus.‡ The heart's sounds may be well heard through the fluid.

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* Even at this advanced period of effusion, alteration of the kind is, however, possible. If a patient with fluid enough in the pleura, when he lies on the back, to render the sound dull to the opposite side of the middle line, be made to lie on the side of the effusion, in the course of a minute or two the sound becomes clear at the mediastinum. I have found this point of considerable utility in certain cases in determining whether there was or was not mediastinal tumour present, in addition to abundant pleural effusion; it cannot, however, be implicitly trusted to [139], as a tumour may gravitate from one side slightly to the other.

† J. Harrison, U. C. H., Females, vol. ix.

‡ Crowhurst, U. C. H., Males, vol. ix., p. 142.

The diaphragm, depressed to a variable amount, carries with it the liver or spleen; with the medastinum, the heart is pushed to the opposite side. The amount of lateral detrusion of the heart is sometimes very remarkable: in cases of left effusion, the organ may pulsate outside the right nipple; and thirty-six hours will sometimes suffice to produce this amount of malposition. The visible and palpable impulse of the organ, and the maximum points of its sounds, especially of the first, prove its change of place. Does the displacement exercise any influence on the quality of the sounds? Most unquestionably it does not, in the great majority of cases; my experience on the point perfectly accords with that of Dr. Stokes, who feels convinced that, even when at its height, the displacement "does not cause any alteration in the natural sounds of the organ." But the rule, I am equally persuaded, is not without its exceptions. Dr. Hope describes the following peculiarity in a case of right lateral detrusion from effusion in the left pleura: "The aorta was felt to pulsate between the second and third right ribs, an inch from the sternum; and here a murmur was heard with the first sound, which has ceased since the heart has been restored to its natural situation by the absorption of the fluid. Is it, therefore, possible that a twist given to the aorta, or pressure of the vessel against the ribs, may be the cause of a murmur under such circumstances?" I once found, for many successive days during the height of left pleural effusion, both sounds of the heart, which was pushed to the right of the sternum, more or less masked by blowing murmurs; these murmurs, when the heart was restored, or very nearly restored, to its natural position, almost completely disappeared,—a fact the more remarkable, as, from the slowness of convalescence, plenty of time had elapsed for the formation of the systolic basic murmur of spanæmia. The diastolic murmur disappeared the more perfectly of the two, and must positively have depended, probably through torsion of the aorta, on the malposition of the organ.* Larrey has related a case in which extreme feebleness of the pulse in the large arteries co-existed with cardiac displacement; there can be no certainty, however, that there was any mutual dependence between the two circumstances.

No matter how copious the effusion, whether acute or chronic, nor how complete the evidences of centrifugal or dilating pressure, signs of centripetal pressure are, as a rule, absolutely wanting,—the trachea, œsophagus and large veins escape serious pressure. Hence the detection of the latter class of signs, in a case of pleuritic effusion, may be accepted as proof of some additional disease, such as tumor or aneurism, within the chest. I have, however, seen a single vein, such as the epigastric, external jugular or mammary, enlarged in calibre in cases of pure effusion. The laws of hydrostatic pressure seem to me readily to explain this exemption of special internal parts from interference.

665. *Stage of Absorption.*—The absorption of pleuritic effusion is effected with results of two different kinds on the form and condition of the chest. The walls may be simply restored to their natural position, or they may sink inwards,—inside, as it were, their natural site. Hence, clinically, we have a period of absorption *without* and *with retraction of the side:* of these the former is infinitely the more favourable.

666. *Without Retraction.*—The visible enlargement and bulging gradually disappear, and with them, but very slowly, the obstructed state of the chest-motions; the natural intercostal hollows, deepened, perhaps, by emaciation, are again felt; friction-fremitus and vocal vibration return,—the former often with greater intensity than at the outset of the disease. The various measurements fall to their natural standards. The percussion-sound gradually recovers its natural clearness, first at the upper, then at the lower, regions; in the latter, indeed, it may long, for weeks, or even months, remain more or less dull

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* H. Morris, U. C. H., Males, vol. vi., pp. 35, 38.

from imperfect expansion of the lung, and accumulation of false membrane in the pleural sac. The respiration-sounds, gradually restored, remain for a variable time weak and harsh or bronchial; friction-sound, mixed or not with pleural pseudo-rhonchus [228], or pulmonary pseudo-crepitation [216], reappears for a variable period; ægophony is sometimes caught passingly for a day or so, but the vocal resonance quickly becomes bronchophonic, or may be null. The heart, with the mediastinum, returns to its natural position, sometimes with singular quickness. I once saw a heart beat in its exact normal site, which, seventy hours only before, I had felt pulsating under the right nipple; absorption so rapid, singularly rare under all circumstances, was here the more remarkable, as the patient was tuberculous, and had had several attacks of profuse hæmoptysis.

667. At this period of the disease very singular signs may temporarily occur. Thus, in a middle aged enfeebled male, undergoing absorption of moderate effusion, with probable acute lung-consolidation conjoined, the following facts were noted. "Tubular percussion-sound in the left infra-axillary region; at the inferior angle of the left scapula the voice is strongly resonant, with a whispered echo after each word, exactly as if the voice were reproduced in whisper under the stethoscope; at this point obscure friction-sound, with thin various-sized rhonchal sounds; the respiration here is hollow, of cavernous quality, especially in expiration. Even with whispered voice the pectoriloquous echo is perfect; though the laryngeal whisper is so low as to be scarcely audible by the ear held close to the patient's mouth." Eleven days later the percussion-sound had become pulmonary in the main at the inferior angle of the scapula; little was heard there in calm breathing; under effort, feeble respiration, mixed with friction-sound and loose rhonchus, became audible; the vocal resonance had lost all the peculiar characters above described, being simply exaggerated, and not hollow in quality.*

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* Fosbury, U. C. H., Males, vol. ix., pp. 350—352.

668. Increased conducting power on the part of the solidified lung substance, combined with dilatation of the bronchi, explains the ringing hollow quality of the respiratory and rhonchal sounds. The vocal whisper within the chest could not have depended on unison-resonance, seeing that it did not synchronise with, but followed, the first resonance; nor upon consonance, for it occurred with various words spoken with not precisely the same pitch: it manifestly depended on echo of the first resonance, which it followed at a certain interval of time.

669. *With Retraction.*—General retraction of the affected side is very much less common than partial depression,—the latter occurring probably about twelve or fifteen times as frequently as the former: general retraction is a process requiring much time for its accomplishment. From the inquiries of M. Woillez, partial depression would appear to be more frequent in front on the right side, behind on the left side. The shoulder, the ribs, and the nipple, fall (in some peculiar cases the shoulder rises, however); the scapula becomes tilted outwards at its inferior angle; the dorsal spine curves laterally, the convexity looking, at least in the majority of cases, towards the sound side; and the ribs undergo distortion, their external planes becoming more or less inferior. This altered condition of the ribs lessens the width of the intercostal spaces; and in cases of very chronic course hypertrophous and osteophytic enlargement of the ribs still farther decreases that width. The chest-surface laterally is unnaturally irregular and uneven; in front it may be flat, smooth, and even slightly concave, the distinction between ribs and interspaces being completely lost to the eye. Rubbing vibration is rarely to be felt. All the chest measurements (those of the side, generally, as well as the partial class) undergo diminution, with the exception of that between the clavicle and nipple, which increases.* The measured chest-play, on the diseased side, may be absolutely, or next to absolutely,

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* This comes of lowering of the nipple,—a more valuable sign on the right than the left side.

dull, while that of the other side exceeds the individual standard of health. The percussion-sound, dull, with marked resistance, inferiorly, acquires the wooden quality at the mid-height of the thorax, and is often tubular at the apex, in front and behind. If dilation of the bronchi supervene, the tubular note may be very extensively discernible. The enlargement of the ribs, just referred to, contributes to give an osteal character to the sound, especially laterally. The signs obtained by auscultation, though less striking than these, are sufficiently important; the respiratory sounds, more or less completely suppressed at the extreme base, are superiorly weak, bronchial or diffused blowing,—months may elapse, after retraction has commenced, before respiration is restored to any extent; friction-sound, of creaking or grating type, may or may not be audible; the voice resounds with morbid intensity, especially at the central parts of the side. The vault of the diaphragm and subjacent viscera are sometimes drawn above their natural level,—an elevation not faithfully indicated by changed position of Harrison's sulcus. The position of the heart varies in at least five different ways:—(*a.*) The organ having slowly or rapidly, gradually or suddenly, retraced its steps, recovers either its natural situation, or the immediate vicinity of this: here is the most common case. (*b.*) It remains in the abnormal position into which it was forced by the effusion, in consequence of the establishment of adhesions. (*c.*) The tractive force may be so powerful at the period of absorption as to *pull* the organ out of its place in the converse direction to that into which it had previously been *pushed:* an occurrence best observable when the right pleural sac has been affected.* (*d.*) It may happen, according to an observation made by Dr. Stokes, that the heart hangs more loosely than natural in the chest, and so falls somewhat to the right or left, as the patient lies on this or that side. (*e.*) Or it may happen, in cases of left effusion, that the heart, originally pushed enormously to the right, subsequently passes to the left, even beyond its natural site,—and still later, regains more precisely its normal position by repassing a very little to the right.*

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* Griffiths, U. C. H., Males, vol. ii. p. 174.

670. The practice of hospitals more especially teaches us that, while pleurisy is among the more common of acute diseases, friction-signs are far from remarkable for frequency of discovery. The following seem the chief reasons of this comparative rarity :—1. Liquid effusion generally occurs with great rapidity; the time during which friction-signs are audible has therefore frequently passed by when patients apply for medical aid ;—2. Friction-signs may exist, but escape attention from their slight degree of development ;—3.—Or from auscultation not being practised immediately over their seat of production ;—4. Or from too long a period being allowed to elapse between successive examinations of the chest ;—5. In cases of absorption of pleuritic effusion, the development of redux-friction sound will be prevented, if the two pleural surfaces be uniformly agglutinated together; because locomotion of the lung is thus prevented ;—6. In all cases of pleuro-pneumonia in which, while the infiltration and enlargement of the lung are sufficiently great to prevent its expansion, resolution of the pleurisy occurs before that of the pneumonia, pleuritic friction-sound (if we believe M. Fournet) cannot be developed. Dr. Stokes has also expressly noticed the rarity of friction-phenomena in pneumonia: "in no case has he found them after hepatisation had formed; and their co-existence with the crepitating râle in the early stages is extremely rare." My observation is somewhat at variance with this statement: friction-sounds are occasionally audible directly over hepatised lung, if the consolidation be confined either to the centre or the periphery of the organ; besides, they are often well marked in front about the nipple, while the signs of hepatisation are perfect behind ;—7. In some, by no means all, cases, friction-phenomena are audible in pleurisy after deep inspiration, when perfectly imperceptible during ordinary breathing. The natural inclination of pleuritic patients to restrain the movements of the chest as much as possible, may then prevent the development of rubbing sound.

671. The physical signs, now described, are those of the disease from the moment of attack to its final term,—in other words, of its acute and chronic forms.†

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* Luckett, U. C. H., Clin. Lect., loc. cit. p. 390.

† Two very rare signs may here be added: perfect *cracked-metal note*, in the first and second interspaces, during the height of effusion and subsequently (Denley, U. C. H., Females, vol. vi., p. 85); and loud rubbing *redux friction-sound*, with four or five jerks in inspiration and expiration, *above the clavicle* (Whiles, U. C. H., Males, vol. vi., p. 122).

Symptoms of Acute Pleurisy.

672. Acute idiopathic pleurisy may or may not commence with rigors; they are rarely strongly marked, and I have never known them of the severity frequent in pneumonia. Rigors, or mere chilliness, followed by heat, generally moderate in amount, and certainly not acrid or burning, may be either actually the first symptom, and anticipate local suffering by some hours or even days, or be preceded for a few hours by the characteristic "stitch in the side." This peculiar pain, dragging and shooting in character, increased by movement, deep inspiration, direct pressure, and percussion, varying in severity from a mere annoyance to a feeling of agony, commonly seated below the nipple, near the antero-lateral attachments of the diaphragm, and under the scapula, rarely extending over the entire half of the chest, sometimes referred solely to the non-inflamed side,* still more rarely limited to the confines of the abdomen, or the abdomen, and this when no physical evidence of local peritonitis exists, felt generally in inspiration more than in expiration, in rare instances solely with the latter act,† is persistent, temporary or intermittent, and sometimes totally disappears, more commonly remits, with the occurrence of effusion.

673. The immediate cause of pleuritic pain is far from clear. Both costal and pulmonary pleuræ may be vascular in the highest degree, the surfaces free from exudation, friction-sound intense, and yet not the least pain or tenderness exist.‡ There may even be strong friction-fremitus from exudation-matter, without a particle of local suffering.§ On the other hand, intense pain may coexist with friction-sound, limited to a surface

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* Luckett, U. C. H., loc. cit. Could this be from coincident pleurodynia?

† Bassett, U. C. H., Females, vol. vii., p. 229.

‡ Wilkinson, U. C. H., Males, vol. ix. p. 284.

§ Humbert, U. C. H., Males, vol. ix., p. 14.

no larger than the palm of the hand.* Obviously friction of inflamed surfaces is not the cause of the pain in question; and the probability is that the suffering depends on intercostal neuralgia or neuritis: still it by no means necessarily reaches its height in persons of nervous temperament. Tenderness under pressure generally exists—traceable sometimes apparently to the pleura itself, sometimes to the intercostal nerves.

674. Increased frequency of breathing is a very constant symptom, perhaps more so even than local pain: it may be unnoticed by the patient, or attended with distressing sense of dyspnœa. The number of respirations very rarely exceeds the third of the number of the heart's pulsations: indeed, I do not remember ever to have observed, in a positively uncomplicated case in the male, a pulse-respiration ratio lower than 3:1.† An enormous amount of effusion may coexist with a ratio of 3. 5: 1. The ratio is more perverted in the sitting than in the lying posture: thus, in a case of copious effusion:—

	P.	R.	Ratio.
Lying . .	122	36	3·39:1
Sitting . .	128	44	2·93:1

It is most important, however, to distinguish dyspnœa from the mere spontaneous hastening of breathing, which sometimes comes of the petulance of pain. The breathing movements, as we have seen, are restrained in amount on the affected side; their increased frequency acts as a sort of compensation.‡ The dyspnœa is generally more marked at the outset, than after effusion has occurred. Cough exists in the great majority of cases, dry (unless there be co-existent bronchitis), short, small, stifled as it were, and frequent; it may, however, be completely absent, though the febrile state be highly marked. During the dry and plastic stages, the patient commonly lies on the sound side or on the back; I have seen exceptional individuals lie by choice on the diseased side, to control movement, and stifle pain, as they assured me: generally speaking, this posture increases pain. After effusion has occurred, the patient commonly lies on the back, on the affected side, or diagonally between both, with the head somewhat raised: he may, however, day after day, while copious effusion exists, lie on the sound side.*

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* Young, U. C. H., Males, vol. ix. p. 294.

† In nervous and hysterical females the ratio may, however, fall lower than this under the influence of painless pleuritic effusion. The temperature of the expired air may fall sensibly below the average of health under these circumstances. Harrison, U. C. H., Females, vol. ix., p. 309.

‡ This restraint of motion on the pleuritic side is a remarkable instance of consensual action, or of motion regulated involuntarily by sensation. It cannot be imitated by the will, and is therefore involuntary; it depends on sensation, and is therefore not reflex.

675. The general symptoms are those of a febrile inflammation. The pulse is frequent, sometimes hard and concentrated; the skin, hot, but not acridly or burningly so, at the outset, becomes moist at the effusion-period. There is but slight prostration of strength; and the cerebral functions are very rarely affected. The urine, small in quantity, is of high specific gravity, deep colour, and strong odour, may be temporarily albuminous, as in other acute affections, has been found in rare instances impregnated with fibrine, and contains oxalates often at the period of convalescence; but in all this there is nothing distinctive. The blood is hyperinotic,—the fibrine varying from 3-5 to 7 per 1000: as a rule, it ranges lower than in pneumonia. The buffy coat is absent from blood drawn in about one fourth of cases.

675* Acute pleurisy is often followed by limited plastic pericarditis, or peritonitis, the latter most common on the right side. Pneumonia is an excessively rare sequence, on the affected side especially; indeed, the effect of fluid pressure is to disgorge the lung of its blood in great measure, as well as of its air. The association of pneumonia and pleurisy proceeding *pari*

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* Harrison, U. C. H., Females, loc. cit.

passu, and each modifying the progress of the other, is not common.* Bronchitis does not often co-arise with acute pleurisy, and is not an obvious effect of it either.

676. The terminations of acute pleurisy are by recovery effected either by resolution or absorption; by lapse into the chronic state; and, under certain circumstances, by death. Death is so rare a result of the disease, when attacking individuals free from organic affections, that I have neither myself (and I have carefully attended to the point, since my attention was first drawn to it, years ago, by M. Louis) lost a patient from pure primary idiopathic pleurisy, with or without effusion, nor known of an occurrence of the kind in the practice of others. And, although, where chronic disease either of the lungs or of other organs pre-existed, death is a more common result, it is still an unusual one. Pleurisy is rarely the immediate cause of the fatal event in phthisis; it is only so by rare accident in chronic bronchitis; and, although both pleuræ suffer in blood-diseases attacking the serous membranes, as for instance, in uræmia and pyohæmia, pleuritis is even here not only an uncommon apparent cause of death, but, when apparently destructive to life, is generally conjoined with pneumonia. I have, proportionately to the rarity of these diseases, found the secondary pleurisy of carcinoma of the thorax and its contents† the most fatal variety.

Symptoms of Chronic Pleurisy.

677. Chronic pleurisy presents itself clinically in three conditions or forms:—(1) With retraction of the side, and the various accompanying physical imperfections already

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* A slight amount of plastic exudation in the pleura is so common in pneumonia that pleuro-pneumonia and pneumonia may, in that sense, be used as convertible terms; but pleurisy of clinical importance, and producing effusion, does not occur in more than about one-eighth of cases of pneumonia.

† Dewing, U. C. H., Males, vol. v., p. 19; Unwin, U. C. H., Males, vol. iii. p. 239.

described: (2) With permanent dilatation of the side by sero-albuminous or purulent fluid (*empyema*): (3) With permanent fistulous opening in the pleura, and discharge of pus from the sac.

678. In the first case of retracted side, the rule is, that the general health is below par,—the individual is thin, incapable of much physical or mental effort, and prone to slight passing attacks of inflammation in the affected side. The breath is rather short, and dyspnœa easily evoked; there is frequent pain in the side. But in exceptional instances, hypertrophy of the fellow lung occurs on an extensive scale, and the patient does not, *quoad* facility of breathing (breathing power is another thing), differ notably from healthy persons.

679. In the second case, of persistent distension with sero-flocculent or purulent fluid (*empyema*),—conditions of the fluid which cannot be distinguished with certainty during life,*—symptoms of more prominent character exist. The patient either lies on the back, or diagonally on the diseased side, with the head slightly raised and bent towards that side, but often acquires the power of lying on the healthy side under the influence of treatment. He rarely suffers from local pain, unless some intercurrent acute inflammatory action arise. His dyspnœa varies in amount; I have never known it seriously pervert the ratio of the pulse to the respiration. The voice is weak; the cough frequent, either dry or attended with expectoration, muco-purulent or purulent. Not only does empyema not give rise in itself to hæmoptysis, but empyema, established in a case of phthisis, appears to a certain extent prophylactic against the hæmoptysis, which is almost an appanage of the latter disease.† Expectoration habitually depends on some

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[*] Bulging of the intercostal spaces was once supposed to occur only where the contents of the pleura were purulent;—a most unquestionable error, as is now commonly understood.

[†] "In sixteen well-marked cases of empyema which I have had under my care within the last two years, for periods of three months and upwards, no

co-existing affection of the lung or bronchi,—it is steady in amount, and rather abundant. But sometimes sudden profuse pouring forth of sero-pus by the bronchi takes place, through perforation of the pulmonary pleura,—or a similar discharge, to the amount of two pints or more, may rapidly take place without any such perforation occurring (at least so far as trust can be put in physical signs, and commonly they are absolutely conclusive on the point), but simply through a metastatic flux from the bronchi. The gurgling rhonchus which occurs in the rare association of a circumscribed collection of pus in the pleura with bronchial fistula may, according to M. Chomel, be distinguished from that of an ordinary tuberculous cavity thus: in the latter, the cavernous rhonchus diminishes in proportion as the examination is made further away from its centre of production; in the case of the pleural cavity, as it may be called, the gurgling produced inferiorly, the most common site of perforation, is propagated upwards, and exists with its full force within a limited extent around.

Œdema of the affected side of the chest is common; it may extend to the corresponding limbs, and half of the abdomen, leaving the other side of the body almost, or completely unaffected. Clubbing of the finger-ends is sometimes strikingly marked. The face is puffy, and semi-transparent, without malar flush; the lips tumid and livid. The external thoracic

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single example of hæmoptysis occurred. But, more than this, empyema established in a phthisical person, seems to be preventive of hæmoptysis. In seven cases of combined tuberculous excavation and empyema, carefully watched and proved as to the diagnosis, either by dissection or by indubitable signs, no spitting of blood had ever occurred. All these seven persons were males. The pressure exercised on the lung by the contents of the pleura, might appear to explain tho fact plausibly enough, especially as the excavations were on the same side as the empyema in six of the seven cases; and I have recently seen a curious exemplification of the apparent influence of excessively acute pleuritic effusion in arresting obstinate hæmoptysis. But, on the other hand, in one case, excavations existed in both lungs."—Author's Report on Phthisis, Br. and For. Med. Rev., Jan. 1849.

veins are not enlarged, the eyes are not prominent, there is no dysphagia, and no stridulous character in the voice or cough: in a word, there are no signs of concentric pressure. The opposite lung becomes hypertrophous and emphysematous; sometimes its bronchi, rarely its parenchyma, inflame.

The pulse is habitually frequent, quick and small; the patient easily falls into a state of syncope; the skin is hot and dry, the febrile action of hectic type, whether the contents of the pleura be pure pus or not; anorexia and insomnia combine with other causes to produce serious emaciation.

680. The terminations of empyema are by death, through slow asthenia; by recovery, through absorption, or through evacuation of the fluid by the bronchi or parietes,—the opening undergoing closure after it has served its purpose; or by lapse into the fistulous form of the chronic disease.

There may, under the latter circumstances, be one or more fistulæ, and the discharge may be trifling in amount, or sufficiently abundant to account in itself for emaciation. In these cases, retraction of the affected side is carried to the extremest amount possible; the measurement between the nipple and the middle line may, as I have seen, fall to one and a quarter inches less on the diseased than the healthy side; and hypertrophy of the opposite lung* reaches its maximum. The symptoms in this variety of the disease are habitually similar to those of the form last described. In rare instances, not only is life prolonged without obvious suffering, but the individual is able, with care, to follow his ordinary pursuits: death in the great majority of cases is the slow result.

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* The lung in this state, instead of receding, when the chest is opened after death, may actually protrude through the opening. I have known the difference in the semi-circular measurements of the sides, equal four inches in a case of the kind. Increase of bulk of the sound lung takes place, with tolerable speed, too; thus (T. Wicks, U. C. H., Males, vol. i., p. 59, 1847,) by measurements made at an interval of eight months, an increase (emaciation having all the while advanced), equalling very nearly two inches in the semicircumference of the side, was substantiated.

681. In the majority of cases the fistulous opening is parietal, sometimes pulmonary, sometimes pulmonary and parietal combined. In rare instances communications form with distant parts by straight or tortuous sinuses, as with the intestine (fæces entering the thorax):* or with a lumbar abscess opening in the groin.† Pus-collections in distant parts sometimes work their way to the pleural sac,—as, for instance, a pyelitic accumulation;‡ or disease of a distant organ, as the stomach, may lead to perforative communication through the diaphragm with the pleura, and produce secondary empyema.§

682. The relations of empyema to other diseases seem to be these. Various diathetic states, the tuberculous, the syphilitic, the purpuric, the cancerous, and that of Bright's disease, appear often to act as its causes, both by leading to, and by preventing direct recovery from, the original acute attack. On the other hand, empyema tends to produce, especially on the right side, disease of the liver, and ascites.‖ A few instances have fallen under my notice where Bright's disease was certainly, as far as the evidence of symptoms went, secondary to empyema,—but whether as an effect or a coincidence seems doubtful.¶ Bright's disease is often, where it exists, the immediate cause of death.

I have not met with any positive evidence that the lung on the unaffected side is peculiarly prone to pneumonia of an uncontrollable character, as asserted by some writers. Hypertrophy of its substance and inflammation of its tubes are the only conditions I have observed with undue frequency.

683. The proportion of cases of acute pleurisy lapsing into the chronic state is unestablished: it is unquestionably small.

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* Krause, Das Empyema, p. 82. † Ibid. p. 80.

‡ Rayer, Mal. des Reins, t. iii.

§ U. C. Museum, Carswell's Drawings.

‖ Wicks, U. C. H., Males, vol. i., p. 59. Griffiths, U. C. H., Males, vol. ii., p. 174.

¶ Griffiths, U. C. H., loc. cit.

684. Empyema is greatly more common in males than in females. Of twenty-two cases of which I have records three only refer to females; Heyfelder* found but one female among twenty empyematous patients; Krause (loc. cit. p. 109) has collected from various authors one hundred and thirty-seven cases, of which ninety-six were in males, eighteen in women, and twenty-three in children. Acute pleurisy is also more common among males, but not by any means to the extent here disclosed in respect of empyema; it would follow then that pleurisy has a greater tendency to degenerate into empyema in males than females,—a result the less to have been anticipated because tuberculisation is more common in the latter sex.†

Diagnosis of Acute and Chronic Pleurisy.

685. The diagnosis of pleurisy is in the majority of cases sufficiently simple; but at all its periods there is a certain chance of confounding it with other diseases.

686. In the dry period, pleurodynia, and intercostal neuralgia, with bronchitis accidentally co-existing, are the affections most easily mistaken for pleuritic seizure. The rules for distinguishing them have already [528] been given.

687. In the plastic stage, pleurisy may, under ordinary circumstances, be distinguished from plastic pericarditis by the respiratory rhythm of its friction-sounds. In those comparatively rare cases, where the heart's action produces friction of its own rhythm within the inflamed pleura, while the pericardium is perfectly free from disease, attention to the rules already laid down [473] will generally remove all difficulty in the diagnosis. The course of events will soon settle the question, should doubt remain in spite of the aid of those

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* Arch. de Médecine, 3ème Série, t. v., p. 59. 1839.

† Cruveilhier asserts that puerperal pleurisy is scarcely less common, at the Maternité, than puerperal peritonitis. Bull. de l'Acad. de Méd. t. i. p. 104. 1836.

rules; if the friction of cardiac rhythm be pericardial, the signs of pericardial effusion will, with almost absolute certainty, quickly follow.

Friction in the peritonæum is, especially at the upper part of the abdomen, with difficulty distinguished, as its rhythm is respiratory, from similar sounds in the lower part of the pleura. Indeed, I know of no positively distinctive character, except the locality of the sound; if this be obviously beyond the confines of the chest, and if there be no such friction in the pleura as to account for rubbing sound being heard under the abdominal walls, as an effect of mere conduction, its origin in the peritonæum must be admitted:* both kinds of sound may or may not be accompanied with friction-fremitus.

688. Pleuritic effusion, when acute, may be confounded with pneumonic solidification. But in cases of effusion, vocal fremitus disappears; in hepatisation, it is maintained at, or raised above, the average of health.† The percussion-sound under the clavicle on the affected side in effusion is tubular or amphoric, unless the whole pleural sac be filled with fluid; such quality of percussion is very rare in hepatisation, and exists over the most, not the least, solidified, parts. In effusion, there is no crepitant rhonchus: nor have I ever found true tubular, sniffling,

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[*] T. Barker, U. C. H., Males, vol. iv., p. 55. Harrison, U. C. H., Females, vol. ix.

[†] This guide is of comparatively little service, when the left side is affected: the vocal fremitus is naturally so weak on that side: luckily for purposes of diagnosis, mal-position of the heart is most readily effected by effusion into the left pleura. The fremitus-sign is almost valueless in persons with very feeble voices, whether naturally so or from disease. Besides, in cases of *very extensive* and *very dense* hepatisation, the fremitus may be impaired in strength, though not to the same extent as with an equal amount of dulness from effusion [45]: vocal fremitus may be caught *along the spine* in some cases of abundant effusion,—and unfortunately in some instances at some distance outwards from the vertebræ. I have often known fremitus feeble and vocal resonance strong over effusion, but have not, to my recollection, met this combination in hepatisation.

metallic respiration: such blowing respiration as frequently occurs in pleuritic effusion is of the diffused variety, deep-seated, except close to the spine,—and slight in amount as compared with the amount of percussion-dulness present. Vocal resonance is either null, ægophonic, or strongly, or weakly bronchophonic; in pneumonia, it is high pitched, metallic, coarse, strongly and snifflingly bronchophonic. If by placing the patient in the prone position, we find the percussion-dulness decrease, the respiration increase, in amount, and friction-sound, before inaudible, appear, the case is positively one of effusion; but if things remain in *statu quo* after such change of position, the case is not necessarily one of hepatisation. In effusion, the heart is displaced, and dulness extends across the mediastinum. It is true, the same extension of dulness beyond the middle line occurs to a very slight amount in some cases of pneumonia; but where this is the fact in hepatisation, change of position does not affect the mediastinal dulness; whereas it does affect this, as already explained, in cases of effusion. The absence or presence of rusty sputa, of acrid heat of skin, and, above all, the state of the pulse-respiration ratio may be appealed to for further aid; an amount of pneumonic solidification, so great as supposed, will produce a ratio of 2 : 1 or 1·5 : 1. I have never known such perversion result from mere effusion, and the ratio *may*, with copious accumulation, fall within the natural limits. Those rare cases of pneumonia, where vocal resonance and respiration-sound are completely deficient, will be considered hereafter.

A much enlarged liver, extending upwards, is distinguished from effusion by the non-protrusion of the lower intercostal spaces (they are, however, more prominent than those of the sound side), by the clear percussion-sound superiorly, and by the tolerably full amount of respiration audible at the posterior base of the chest. The interlobular fissure of a merely enlarged liver maintains its natural relationship to the middle line of the body; that of a liver pushed down by pleural effusion (as

Dr. Stokes has shown) lies at an unnatural angle with this. If an enlarged liver displace the heart, it does so in an upward, not, as pleuritic fluid does, in a sideward direction. Deep inspiration increases the area of clear percussion-sound inferiorly, and also that of vocal fremitus, in cases of hepatic enlargement;—it exercises no such influence when dulness depends on effusion.

The spleen, when sufficiently enlarged to increase the width of the left base of the thorax, raises the heart; does not, like effusion, push it aside; extends far into the lumbar region, and forwards to, or beyond, the middle line of the abdomen, with a firm, smooth, or nodulated, surface; affects but very slightly the amount of respiration at the posterior base of the chest, and causes no protrusion of the intercostal spaces. Splenic blowing murmur may sometimes be caught. Leucohæmia, if present, will confirm the inference, otherwise deduced, as to the existence of a certain kind of splenic enlargement; but a natural state of the white blood-corpuscles will not exclude the idea of other enlargements.

The distinctive marks of hydro-thorax, intra-thoracic tumor, and cancerous infiltration of the lung, will be described with those diseases.

Tubercle, in its ordinary seat, at the upper regions, cannot be confounded with effusion, which accumulates below. If the entire lung be solid from tuberculous disease, softening and excavation signs exist superiorly, and the progress of the signs is from above downwards; in effusion, they advance from below upwards. Dulness is never as absolute in phthisis as in effusion; some amount of resilience of the parietes remains too. Mensuration proves the existence of diminished bulk in phthisis,* of increased width in effusion. The heart, if displaced in tuberculous disease of the left lung, is carried upwards; in effusion of the left pleura, to the right side: in tuberculous disease of the right lung, the heart may be drawn to the same side; in right effusion, the heart is pushed to the left. Tuberculous disease of an *entire* lung does not exist without implication of its fellow; any amount of effusion may exist in one pleura, the other remaining unaffected. In effusion the respiration is null or weak, distant and diffused blowing; in phthisis, superficial, of various qualities, and attended with rhonchi.

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* In acute tuberculisation with attendant pneumonia, however, this measurement may be increased. Hodson, U. C. H., Males, vol. ix., p. 17.

689. The signs of the absorption period of pleurisy, in active progress, cannot be confounded with those of any other disease. Pleural pseudo-rhonchus and pseudo-crepitation in the lung, from unfolding of its shrunken substance, can with very little care be distinguished from true pneumonic crepitation, either primary or redux.

690. Pleurisy, at the period of absorption with retraction, can only be mistaken for affections attended, like itself, with diminished bulk of the affected side,—viz., tubercle, chronic pneumonia, so-called "cirrhosis" of the lung, and infiltrated cancer. Where tubercle diminishes the measurements of the side, it does so mainly superiorly, pleurisy mainly inferiorly: the respiration-movement is greater in tubercle than in chronic pleurisy with retracted side. In tubercle, both lungs are diseased; in chronic pleurisy the organ of the unaffected side may grow extra-healthy, hypertrophous. The signs of softening of the lung are, of course, wanting in the pleural affection. The distinctive signs from the other diseases named, are given with the histories of these.

Treatment of Acute Pleurisy.

691. In the acute disease, especially if the febrile action be of sthenic character, venesection to an amount proportioned to the urgency of the symptoms, and the strength of the individual, should be employed; from a healthy male adult, from twelve to

twenty ounces of blood may be taken with propriety. Local depletion by leeches, or better by cupping, should follow this, if the pleuritic pain continue; and in cases of moderate severity, should be the only mode of blood-letting employed. I have not observed any more favourable results in cases where blood-letting was pushed to extremes, than where used in moderation: the risk of anæmia and protracted convalescence should not lightly be incurred; and, on the other hand, the time for absolutely arresting the disease in its course has generally passed, when the patient comes under observation.*

Next in order, not inferior in importance, to blood-letting (evacuation of the bowels having been quickly effected) comes mercurialisation—to a slight amount—of the system: the more rapidly this influence is produced, the better: and hence, for the first six hours, small doses of calomel and opium, (a grain and a half of the former to a sixth of a grain of the latter, or more, if the pain continue acute) should be given every half-hour, while mercurial ointment is rubbed, every fourth hour, into the affected side, especially near the axilla. A patient thus treated should be watched from hour to hour almost, and the moment mercurial action exhibits itself, the mineral should be stopped; ptyalism is not the object to be attained. After some twelve or twenty-four hours, small quantities of tartar emetic, or of

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* A boy (Henry Falkness) was admitted in the evening of January 10, 1851 (U. C. H.), with acute pain in the side, and dyspnœa. My clinical assistant, Mr. G. Webster, finding marked friction-sound on examination, ordered twenty leeches to the spot. The next day I could not detect a vestige of friction; effusion-signs neither occurred then, nor at any time (the boy leaving, perfectly well, ten days after admission,—he was detained thus long for purposes of observation). About the fourth day a shade of grazing friction-sound was caught in the old spot. Were exudation and effusion absolutely prevented here by treatment; or was the case one naturally inclined to stop short at the *dry* stage? The friction-sound formerly appeared to me to have been almost too intense for the latter hypothesis; but the case referred to in a previous page (p. 352, note) shows that friction of the most marked type may exist without exudation-matter.

James's powder, may be given, in combination with the mercurial, if (what is rare, on the plan described), this have been required so long to produce its special mode of action. Small doses of opium and ipecacuanha at night allay cough and irritation.

As soon as depletion has been pushed as far as is intended, and the febrile action has more or less completely yielded, a large blister may be applied to the affected side, but not precisely to the mainly affected part; its influence over yet lingering pain is sometimes most remarkable, and it probably disgorges the sub-pleural vessels, by causing effusion from them, and by loading those of the skin. The application of a blister, directly over the seat of friction-sound, sometimes, it is true, removes this within a few hours, but too rapidly to justify the idea of absorption having occurred; probably a small quantity of fluid, thrown out beside and within the layers of plastic lymph, renders their collision noiseless.*

If, in spite of these measures, fluid continue to accumulate—or if that already thrown out remains stationary—a succession of flying blisters, each of them not kept on for more than six hours—and friction with ioduretted liniments, should be employed externally, while diuretics are steadily administered internally; nitrate, acetate, and bitartrate of potass, squill, nitric æther, juniper, infusion of digitalis, particularly if the pulse remain frequent, are the agents of this class commonly used. I believe the compound tincture of iodine, in scruple doses, freely diluted, to be a valuable medicine at this juncture. The action of any of these medicines is facilitated by the exhibition of blue pill, digitalis, and squill, every night, or every second night.

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* The late Dr. Macartney, of Dublin, taught that flogging sometimes produced pleurisy in schoolboys,—an opinion they would doubtless wish were generally made known to schoolmasters: if the fact be so, it would illustrate the local effect of blisters referred to in the text. Upon the more or less close proximity of a blister to an inflamed surface will probably depend its exercising an *antagonistic* or *sympathetic* effect.

The diet should be low, and if there be effusion to any amount, much drink should be abstained from; enough will be taken with the saline diuretics employed.

692. Is paracentesis advisable or admissible, while the disease is acute?

693. I can find no positive evidence in favour of the operation as a curative measure. If the disease be simple, non-diathetic, the treatment described will remove effusion in the great majority of cases,—in the few that remain, surgical interference does not seem to be warranted by recorded results. Sédillot, a warm advocate of paracentesis under a variety of circumstances, has collected cases showing that "in the acute stage, the operation produces an excitement capable of aggravating all the symptoms, causes more or less abundant hæmorrhage, and accelerates death." M. Gendrin, at one time uniformly operating in cases of acute effusion, was forced by the "constant death of his patients" to relinquish the practice.* Dr. Bowditch,† in a recent essay, advocating early paracentesis, gives but one case in point. Here, a woman labouring under acute pleurisy, treated up to the seventh day by "rest, demulcents and a sinapism to the side," was operated upon on that day. Five days later the fluid had re-accumulated quite as abundantly as at first. On the thirty-second day of the operation, "all the symptoms were gone, save an occasional pain in the side, and some dyspnœa,—accounted for by her pregnancy perhaps." Can any reasonable doubt be entertained, that results, at the least, as favourable as these, might have been obtained by ordinary treatment? the fluid discharged was transparent and viscous: and no friction-sound is mentioned from first to last.

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* Sédillot, de l'Empyème, pp. 127, 83.

† On Paracentesis Thoracis, p. 22, Boston, U. S.

694. As a palliative measure, where pleurisy is connected with tuberculous or other diathetic disease, the operation seems occasionally to have relieved in the acute stage,—more frequently to have placed the patient in a worse position than before. Writers speak of its necessity, where asphyxia is threatened by the copiousness of effusion; such form of danger I have, however, never witnessed in the acute stage of the simple disease.

695. If abundant effusion on one side be associated with single or double pneumonia and general bronchitis, and, depletion having been carried as far as permissible, urgent dyspnœa continues, temporary relief at least will be obtained by paracentesis, and time thus gained for the action of remedies. But experience is wanting.

Treatment of Chronic Pleurisy.

696. The chronic disease presents itself practically under three main conditions: first, the thorax is retracted, the lung incapable, the side painful, the general health impaired; secondly, the side is retracted, its wall fistulous, and a more or less profuse discharge of pus is habitual; thirdly, the side is permanently distended with fluid (*empyema*).

697. In the first case, art can do no more than palliate symptoms as they arise, and support the strength on general principles. Fortunately in some such cases all local inconvenience ceases after a time, and moderately good, if not robust, health is obtained. The wasting suppuration in the second case points to the necessity of tonic treatment: quinine, iron, and cod-liver oil, are essential to the support of the individual. Efforts may be made, by injecting the sac with slightly stimulant fluids, to alter the character of its secreting surface, and perhaps even induce its sides to cohere: ioduretted fluids are the best suited for the purpose.

698. In the management of the third condition, that of

empyema, three indications present themselves: (1) the control of febrile action; (2) the promotion of absorption; (3) the support of the general health.

699. (1.) If antiphlogistic measures have not been put in force with sufficient energy during the acute stage; if febrile action, accompanied or not with local pain, exist; and if the constitutional powers appear not to be severely depressed,—general bleeding, to a limited extent, may be cautiously had recourse to. But it is easy to do mischief by venesection; even under the circumstances supposed, an amount of depression will occasionally follow moderate loss of blood from a vein, which may not easily be recovered from.

The application of some six or eight leeches once or twice a week to the side, for a week or two, is not open to the same objection; and even where local pain and febrile action are totally absent, much benefit may be obtained by the practice, provided the effusion be not of old standing. Or some three or four ounces of blood may be drawn on each occasion by cupping.

Is mercury advisable at this period of the disease? Certainly not, if it have already been used in the earlier stages; certainly not, if there be much wasting of the system, or if there be reason to fear that the fluid in the pleura is purulent. Under other circumstances, a cautious trial of mercury by the mouth and by inunction ought to be made.

700. (2.) Should the disease not yield to these measures, the promotion of absorption is to be further attempted by the use of medicines acting on the secretions; or the use of such medicines may be commenced, while the foregoing plan is pursued.

Iodide of potassium and liquor potassæ in full quantity, the former in doses of from five to ten grains, the latter in doses of from fifteen minims to a drachm, thrice daily, especially if care be taken that the stomach is empty when they are swallowed, sometimes very obviously reduce pleuritic effusions. But the total failure of iodide of potassium, even when pushed to repeated iodism, is unfortunately far from uncommon.*

Diuretics should be exhibited in various forms: the salts of potass, especially the bitartrate and acetate (the former in half-drachm doses, or upwards), and infusion of digitalis, may be commenced with. Digitalis both acts more efficaciously as a diuretic, when administered in infusion, and appears less prone to produce those poisonous effects which occasionally result from its accumulative action. The compound tincture of iodine, largely diluted, is now, as earlier, a very valuable agent of this class. The sustained use of *purgatives* is perhaps deserving of trial, if diuretics fail; but their action, being of a more depressing kind, requires to be carefully watched. They should be very cautiously exhibited when the lungs are tuberculous, either because the intestinal walls already contain tubercle, which may thereby be more speedily brought into the stage of softening, and induce ulcerative destruction, with its consequences, of the mucous tunic; or because the irritation of the bowel in a person already phthisical accelerates the deposition of tubercle there. I have seen more than one case in which uncontrollable diarrhœa, brought into existence under the former circumstances, evidently hastened the patient's dissolution. On the other hand, dyspnœa, which has resisted various other measures for its relief, may occasionally be removed or materially relieved, and this almost instantaneously, by an active hydragogue. I have observed this from the exhibition of elaterium, even where no manifest change in the thoracic physical signs followed. *Diaphoretics* may be employed, particularly if there be occasional febrile action; but in neither these medicines nor in *expectorants* as promotive of absorption, can any confidence be justly placed. *Counter-irritation* of the affected side has been employed in every variety, from the mildest to the most severe. The only contra-indication to the use of agents of this class, is the presence of fever. No matter what precautions be taken, their very common tendency is to increase this, where it exists, and hence to promote a condition extremely unfavourable to the advancement of absorption. "First remove feverish action, and then counter-irritate," is a maxim of primary importance. The application of *blisters* exercises a very manifest influence on effusions, when had recourse to with the caution just stated. They should be repeated frequently, made of large size, applied in different situations, not kept on longer than is just sufficient to produce vesication, and every means used to secure rapid healing of the blistered surface.* Employed in this way for some time—say a fortnight—blisters are often most signally beneficial; and I am the more anxious to state emphatically their utility in these cases, because the incredulity as to their efficacy in *acute* parenchymatous and membranous inflammations, now arising among the profession, might possibly be extended to their action in *chronic* maladies of the kind at present under consideration. Should any objection to the use of cantharides exist in particular cases, the tartar-emetic ointment, or a Burgundy-pitch plaster, sprinkled with tartarised antimony, may be substituted; but these are much less advantageous applications. Issues, setons, moxas, and the actual cautery, have all had their advocates and employers in the very advanced stages of the disease. The moxa is probably the most advisable of these, both positively and negatively, and may be had recourse to when the use of blisters has failed to affect the disease. The moxa will be comparatively most beneficial when the effusion is of very long standing, and attended probably with anatomical change in the bones and ligaments.

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* Crowhurst, U. C. H., Males, vol. x.

701. (3.) When the disease has become decidedly chronic, the general health is to be sustained by a succulent non-stimulant diet. Strong broths, meats of easy digestion, jellies, &c., may be taken with a degree of freedom proportional to their

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* Of all such means, dressing with cotton wool is the best.

observed effects; and to this increasingly liberal diet may be added the use of cod-liver oil and bitter tonics, combined with small doses of nitro-muriatic acid. If these be well borne, the various preparations of iron, especially the syrup of the iodide, should be employed. There is decidedly a stage of the malady, or, perhaps, rather a condition of the organism generally, in which, although some feverish action be present, the patient will be benefitted by tonics.

Change of air sometimes exercises a very manifest influence on the condition of patients with empyema,—primarily, it would appear, on the general health, and, through this, on the effusion. The mere fact of change almost seems to suffice; for there may be no positive superiority in the new atmosphere, in which the improvement occurs, to that for which it has been exchanged.

702. The modes of treatment now described, sometimes fail altogether. The effusion either remains stationary, or increases, and evidently must destroy the patient in the end. Under these circumstances, a resource is still left in the operation of *paracentesis*,—a procedure which, no matter how divided opinion may be respecting its general feasibility, has assuredly been sufficiently often either completely successful, or productive of marked improvement, to justify its being numbered among the valuable gifts of surgery. This is a vague estimate of its utility, it may be alleged; but, unfortunately, such vagueness of expression is unavoidable. In truth, there are no existing data from which precise inferences may be drawn as to the success of this measure, whether considered generally, or in reference to its performance in particular states of the constitution. And this, because too many observers have contented themselves with merely ascertaining the existence of pleuritic effusion in patients operated on, without systematically inquiring into the conditions of the other organs, and, above all, substantiating the presence or absence of pulmonary tuberculisation. How can a correct general inference be obtained, when patients are clubbed

together, who have simple chronic pleurisy, or this combined with carious destruction of the ribs; who have or have not serious organic disease in other regions of the body; whose lungs are sound, or the seat of active tuberculous disorganisation? During the discussion on the subject of empyema, which took place some years since at the Parisian Academy of Medicine, M. Louis well pointed out the importance of taking the general condition of the patient into consideration, when we attempt to estimate the chances of recovery from pleuritic effusion, either with or without paracentesis. And if we admit Laennec to have been justified in advocating the performance of the operation in the case of tuberculous patients, because even in them it may be the means of somewhat prolonging life, we must aver that the plea on which he based this advocacy,—that a natural cure of tuberculous cavities does sometimes arise,—was of rather a visionary character. I strongly doubt that such cure has ever arisen in persons having superadded empyema.

703. The quality of the effusion appears to influence the prognosis of the operation. Where this has been purulent from the first, success has more rarely ensued than when sero-albuminous. But cases have terminated favourably, wherein the fluid was, if descriptions of physical characters may be trusted to, really purulent. In respect of quantity, the less abundant the effusion, it is said, the stronger the chances of success. Recovery has, nevertheless, followed when the fluid measured several quarts.

Empyema running a chronic course from the outset, is generally indicative of tuberculous disease of the lungs, and is so far an unfavourable species for operation. Yet, inasmuch as the exudation-matter in such cases is very frequently friable and imperfectly plastic, and the lung therefore less firmly compressed and bound down, than when the solid exudation possesses the opposite qualities, one condition conducive to successful issue—facility of expansion on the part of the lung—is secured.

But this element of success, it is almost needless to add, is more than counterbalanced by the constitutional state of the patient.

A notion of the probable result of the operation may be had from the condition of the functions in general; if the conservative functions, digestion and nutrition especially, be only moderately impaired, the chances of the patient's recovery are much stronger than under the contrary circumstances. The operation has been much more successful in young persons than in those of mature years; and somewhat more so on the left, than the right, side of the chest.

704. The determination of the period of the disease most favourable for operating, is a point of very serious importance. Experience proves, as might have been anticipated, that when performed at an advanced period, paracentesis is rarely curative, often baneful: the local changes which have then had time to arise in the pleura, pernicious as these are, are even less subversive of success than the deep constitutional distress entailed by the disease. And yet, to this period, puncture of the chest is often, I might almost say commonly, postponed; it can hardly be matter of surprise, that in some such cases it has appeared to do little more than hasten death. There can be no question that the fitting time for operation has come, when a tendency, insuperable by medical means, exists either to increase or to non-absorption of the fluid. The practical difficulty is to determine the precise period at which such tendency may be considered to be developed. On the one hand, perseverance in a given mode of treatment has sometimes proved successful, when everything seemed to foretell its probable inefficacy; and on the other, valuable time may be frittered away, and the period at which paracentesis affords real chance of permanent relief but too easily suffered to pass by, from the procrastination caused by a very natural anxiety to give more gentle measures a full trial. A thorough consideration of the whole history of the case, as also of the existing local and

constitutional states, is required to enable the practitioner to seize the propitious moment for surgical interference. This difficulty is unfortunately not confined to the operation for empyema.

It appears to me scarcely philosophical to fix a particular day of the disease beyond which delay ceases to be permissible: the actual indications for the operation are as strong in one person at the end of a fortnight as in another after the lapse of three weeks. But I certainly believe Hippocrates was right in his precept that paracentesis should not be performed before the fifteenth day of effusion.

704. Let us suppose the operation to be determined on; what is the best plan of performing it? As a measure of precaution, especially if any doubt exist as to the precise nature of the case, a grooved needle may, as suggested by the late Dr. T. Davies, be introduced into the pleura: if the expected fluid appear, the operation may be continued with a trochar or a bistoury. The trochar is certainly the more advisable instrument, if the intention be to evacuate the fluid by degrees,—an intention which we believe Laennec to have been right in commending, when the patient is so debilitated as to justify an apprehension that the complete discharge of the liquid might be followed by dangerous syncope; and when paracentesis is merely performed as a palliative in advanced tuberculous cases. Even when the intention is to remove at once as much as possible of the fluid, the results have on the whole been most favourable with the trochar.

705. The first point to be ascertained in selecting the place for the performance of the operation, is the adherence or non-adherence of the lung to the chest, and, in the former case, if possible, the precise limits of the adhesions. It is scarcely necessary to say, that the site of these must be carefully eschewed. When the entire side is free from adhesion, the common advice of surgical writers has been, that the opening be made at the most dependent part of the antero-lateral part of the chest. But

undiscriminating attention to this advice, given with the sole view of securing free egress for the pleural fluid, has occasionally led to perforation of the diaphragm and abdominal viscera; even the kidney has been extensively wounded by operators whose practice is to adhere too scrupulously to such injunctions. Laennec recommended the fifth interspace, a little in front of the digitations of the serratus magnus muscle, as the fittest site for puncture. The importance of opening the fifth instead of a lower space, has been justly insisted on by Drs. Townsend and Stokes, who draw a fair analogical argument in favour of high puncture from the elevated position in which the discharge commonly occurs, when spontaneous or preceded by the formation of abscess: the opening has, then, sometimes taken place even above the clavicle. If the ribs cannot be clearly distinguished on account of the presence of marked œdema, some difficulty may arise in fixing upon the spot for incision. Steady and continued pressure with the pulps of the fingers will sometimes enable the operator to discover the edges of the bones, in cases where this would on first view have appeared altogether impossible.

706. The place for operation having been duly determined on, a minute incision should be made through the skin for the introduction of the point of the trochar, if this be the instrument chosen for operating with. If the bistoury be selected, an incision of about two inches in length is to be made through the integuments, and the intercostal muscles carefully divided to the same extent. The cutting instrument should not be carried too close to the borders of the ribs, lest the corresponding branches of the intercostal artery be wounded; this statement applies especially to the lower border of the upper of each pair of ribs, along which the superior, and of the two the much larger, twig runs. The division of the costal pleura itself is by some recommended to be performed with a lancet, but such caution seems useless. When the flow of matter announces the pleura to have been divided, the opening in that

membrane should be enlarged sufficiently to give free outlet to the contained fluid.

707. If the operator have been unfortunate enough to incise the pleura over the site of firm, thick, and strongly adherent pseudo-membrane, and in consequence no fluid appear, the line of conduct to be further pursued must vary with his confidence in the correctness of his diagnosis in other respects. If he still remain satisfied of the presence of liquid effusion, the attempt to evacuate this may be proceeded with; if he be apprehensive of having altogether erred, the obvious course is to dress the wound, and avert, as effectually as possible, the ill effects of the unnecessary suffering to which the patient has submitted. In the former case, surgeons have attempted to tear asunder the pleural adhesions, a procedure utterly unjustifiable: the only admissible modes of acting are, to enlarge the existing opening with the bistoury, or to make a new one. The opening may be enlarged, if the free border of the membranous mass interfering with the escape of the fluid be ascertained to be perfectly close to the angle of the existing wound; under other circumstances, a new incision must be made.

708. Two other methods of opening the chest date so far back as the time of Hippocrates. These are, 1, *perforation of a rib;* and, 2, the *formation of an eschar, either by the actual or potential cautery.*

709. The plan of perforating a rib with a trephine constructed for the purpose, fallen into disuse since Hippocrates, has been recently revived by M. Reybard. The object of this surgeon, in recurring to this antiquated practice, was to ensure a solid support for a canula, which he proposed leaving in the pleura, as a sort of drain for, or, at least, means of removing at will, any new accumulation which might arise. A case thus treated, previously operated on in the ordinary way, terminated favourably; and neither caries nor necrosis of the bone appear to have complicated its progress. From this single case, it is,

however, impossible to draw any safe inference as to the general expediency of the plan.*

710. Modern surgery repudiates the idea of opening the chest by an eschar produced with the actual cautery; and we can imagine no advantage gained by substituting caustic potass for the trochar or bistoury, while numerous very serious objections to such a procedure manifestly exist. Nevertheless, in cases where an external abscess has formed, and in these alone, the opening in the skin may, if the patient be excessively timorous and impressed with a dread of cutting instruments, be made with a piece of caustic potass.

711. Some writers lay very great stress upon the importance of preventing the occurrence of pneumothorax; and there can be little question that the entry of air through the wound is a circumstance which it would be well, if possible, to avert. But the efficacy of any plan devised for its prevention is extremely questionable. Thus the modifications of the common canula, constructed for the purpose of obviating the occurrence of pneumothorax, have some invariably, others occasionally, been found failures. The proposal to puncture the chest under water is worth trial. It seems, in truth, well to prevent the ingress of air into the pleural cavity, if possible; and this because the existence of pneumothorax must interfere with the expansion of the lung; and the contact of air with the diseased surface is likely to entail increase of inflammation and decomposition of the fluid retained or re-secreted by the pleura. Nevertheless, the occurrence of pneumothorax does not by any means appear to be a matter of such importance, as considerations of this kind might lead us to expect. In numerous instances, where the physical signs of pneumothorax were distinctly detected after the operation, recovery has ensued.

712. Laennec appears to me, as already stated, to have

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* Gaz. Méd. de Paris, Janv., 1841.

correctly pointed out the description of cases in which it may be advisable to evacuate the contents of the chest, by degrees, and upon successive occasions. Under other circumstances, experience shows that no apprehension need be entertained of ill effects following complete evacuation. Speculative reasons may and have been, by various persons, adduced in numbers in favour of each mode of evacuation; the important point to remember is, that experience is in favour of as complete evacuation as possible. Curiously enough, many of the advocates of successive evacuations are at the same time energetic in denouncing the ill effects of pneumothorax; how they reconcile to themselves the recommendation of a mode of operating which doubles, trebles, quadruples, or even still further increases the chances of the entry of air, does not very clearly appear.

713. The proposal to ensure the removal of the entire collection of fluid, by drawing off with a syringe any portion of it which gravitates below the opening, is as old as Scultetus. Mr. Jowett and others have proposed modifications of the syringe employed by that surgeon. There does not appear to be any reasonable objection to the employment of these instruments, if they be dexterously introduced and managed: and they may, in some cases, be decidedly useful. Laennec proposed the use of a cupping-glass and exhausting syringe, with the view of drawing off the remains of the fluid, and facilitating the expansion of the lung.

714. Is it advisable, under all circumstances, to close the wound after the operation? If a fine trochar have been employed, the orifice closes without artificial help, and scarcely requires dressing of any kind. If the bistoury have been used, and the contents of the pleura have been completely evacuated, there can scarcely be any motive for leaving the orifice open. If the evacuation have been only partial, still it is better to close the opening than to leave a canula or tent in it, as the constant renewal of the air in the pleura is decidedly detrimental, and

may, unlike entry of air at the first, be prevented by immediate closure of the wound. If, however, any species of canula were devised which would admit of the egress of fluid, and successfully oppose the ingress of air, this objection will cease to exist; and the facility afforded by such an instrument, for the removal of any newly-secreted fluid, will be an important motive for its employment.

715. The changes induced by the operation are, or may be, of two kinds; *physiological* and *pathological*. The former, which are local and general, are as follows: the diminution of the contents of the chest is, of course, attended with decrease in the dimensions of the affected side. In a case observed by Dr. Townsend, the semi-circumference, which reached 16½ inches before the operation, had fallen to 13¾ inches on the ninth day after it. The approximation of the pleural surfaces is rendered manifest, sometimes at a very early period, by the occurrence of friction-phenomena; the patient is frequently enabled to lie on either side on the day, or even a few hours, after the operation: his dyspnœa is immediately relieved; the state of anxiety under which he laboured disappears; and a sound sleep, the first, possibly, enjoyed for weeks, commonly follows the evacuation. Even in cases eventually terminating fatally, and terminating fatally through the influence of the operation, its immediate results may be of this favourable character.

716. Of the pathological effects, as they may be termed, of the operation, pneumothorax is the most inevitable: the question of its influence on the ultimate issue of the case has already been referred to. The continuance of secretion from the pleural surface for a greater or less period, and hence the constant renewal of empyema, is of very common occurrence. In rare instances, the characters of the newly secreted fluid remain those of that originally evacuated; in the great majority, they change, the general tendency of the change being to the purulent character. The alteration from the almost purely serous appearance to the purulent is sometimes accomplished in twenty-four hours.

When the fluid has been originally more or less completely formed of pus, but of a laudable kind, its conversion into a purulent matter, of bad quality and fetid smell, is not unusually observed. Under these circumstances, the injection of warm water or some other unirritating fluid becomes advisable. Should there be much appearance of putrescency, a small quantity of very dilute liquor sodæ chlorinatæ ought to be added.

717. Although the notion of giving tone to, and diminishing the irritation of, the diseased pleural surface by injections of tonic and alterative preparations, seems, on first view, of questionable justness; and although the practice has certainly not been attended with sufficiently favourable results to warrant its general employment; yet a fair number of cases are recorded showing that ioduretted solutions, at least, may, under certain circumstances, not only be injected with impunity, but with the effect of completely curing empyema of long standing. M. Boinet, in particular, relates cases of this fortunate issue.*

718. In the ordinary course of things, when the case is destined to end by the patient's restoration to health, the wound or wounds in the thoracic walls gradually close, and cicatrisation is perfect within a short period; but in some cases, the opening, instead of closing, acquires the characters of a fistula, which it retains for a variable period, and daily gives issue to more or less pus.

719. When spontaneous perforation of the costal pleura has occurred, and a portion of the contents of the pleura escaped through the opening into the subcutaneous cellular membrane, forming an abscess there, this should be opened without delay, in order to prevent the occurrence of sinuses, and burrowing: so

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* Arch. de Médecine, Mai, 1853. The proportions of tincture of iodine and iodide of potassium varied, in the cases related, between ten and fifty of the former, and one and four of the latter, to a hundred parts of distilled water. Severe iodism may follow the use of the stronger preparation. The injections may be repeated once, twice, or thrice a week, according to circumstances.

well established is this point, that the operation is under these circumstances termed *empyema of necessity*. The prognosis of paracentesis, as far as the operation goes, is, in this exigency, favourable above the average.

720. Cases of *double* empyema are, as a general rule, unfit for operation, unless evacuation be rendered necessary for the prevention of asphyxia. Should particular circumstances arise, under which paracentesis might on other grounds become admissible, an interval of time, varying in length with the condition of the patient, must be allowed to elapse between the two operations.

VARIETIES OF PLEURISY.

721. The varieties of pleurisy may be arranged according to the following plan:

I. *Idiopathic.*

Seat.	Single; double. Unilocular; bilocular; multilocular. Circumscribed; costal; pulmonary; phrenic; mediastinal; interlobar.
Course.	Obvious. Latent. Pulsating.

II. *Secondary.*

Attending general diseases.		Typhoid, typhus, eruptive, and puerperal fevers.
Attending diathetic diseases.		Scrofula; Bright's disease; rheumatism; gout; scurvy; purpura; pyohæmia.
Caused by adjacent irritation.	In the lung.	Pneumonia, abscess; tubercle; cancer; gangrene; hydatids.
	The adjoining organs.	Stomach; liver; spleen; pericardium; mamma.
Traumatic.		Contusions, wounds, and surgical operations in the chest-wall, perforative and non-perforative, with or without entry of foreign bodies; fracture of ribs or clavicle; hæmothorax.

Perforative	Through lung.	Tubercle; abscess; cancer; gangrene; vesicular emphysema; hydatids; foreign bodies from bronchi.
	Through diaphragm.	Cancer of stomach, œsophagus and liver; hydatids of liver; chronic pyelitis.

722. It appears from this scheme that, under a multitude of different circumstances, pleurisy is a disease for which the physician should be on the watch. The peculiarities of some of these varieties have already been spoken of; certain other varieties will be considered with the diseases producing them: in the present place, a few only of the number will be briefly dwelt on.

Circumscribed Pleurisy.

723. The portion of pleura inflamed may be limited: the disease is then said to be *circumscribed.* When the *phrenic* surface is solely, or specially, affected, the pain is unusually severe; orthopnœa is frequent,—attended even with bending forwards of the trunk; the cough is strikingly paroxysmal; while hiccup, nausea, vomiting, delirium, excess of upper costal breathing, jaundice (if the disease be on the right side), and, occasionally, *risus sardonicus,* combine to impress somewhat distinctive characters on the disease in this locality.

724. Inflammation of the *mediastinal* pleura is often accompanied with serous infiltration of the cellular tissue of the mediastinum itself; *pseudo-rhonchus* will then be heard along the sternum.

725. Pleuritic fluid accumulation, confined by adhesions between the lobes of a lung (*inter-lobar* pleurisy), may simulate a solid mass in the pleura or in the lung itself, or an aneurism. Dull percussion-sound, local bulging, weak or bronchial respiration, with vocal resonance, may exist in all these cases. The pleuritic accumulation lies in the line of the inter-lobar fissure of the lung; the voice may have an ægophonic twang on its confines; there is no vocal fremitus over it; there is neither impulse

nor murmur; and the affection has a past history different from the other diseases named. Similar local collections may form with a boundary of adhesions in any part of the pleural surface, and several such may co-exist, forming sacs perfectly independent of, or communicating with, each other,—whence bilocular or multilocular empyema. The adhesions, forming the walls of these loculi, of course unite portions of lung-substance to the surface, and so give rise to various modifications in the physical signs. The general character of these modifications is, that wherever adhesions exist, blowing respiration is heard; and if a portion of lung, of any size, even though condensed, be agglutinated to the surface, the percussion-sound will be less dull than directly over the fluid; the state of vocal resonance varies. I have, on the right side, found the vocal fremitus perfectly retained over such portions of lung.

726. Individuals, who have been the subjects of extensive pleuritic agglutination, consequent on general effusion, undergoing subsequent attacks of pleural inflammation, present the signs of these multilocular collections in perfection. Isolated patches of surface, simultaneously or successively, and in positions the most unconnected, furnish the signs of exudation, effusion, and absorption, while intervening islets of the chest-wall remain perfectly free from any such signs. In one remarkable case of this kind, four successive attacks of pleurisy, occurring within a period of five months, were thus characterised.

Latent Pleurisy.

727. Pleurisy is said to be latent, when it runs its course without producing decided subjective symptoms,—when there are neither local pain, cough, dyspnœa, nor febrile action. In cases of this kind, effusion may have reached to the clavicle, and driven the heart greatly out of its place, and yet the patient remain utterly unaware that his chest is the seat of disease. He seeks advice, either from a vague consciousness that he is

not in his usual health, or for some ailment totally unconnected with the thorax. The physical signs reveal the true condition of things. The necessary treatment, once the disease is detected, is not modified in any important point (except that blood-letting need not be carried at all so far) by this latency of course; the great difficulty often consists in persuading the patient that there is really anything of a serious character to treat. I have repeatedly known persons with copious effusion of this kind follow their usual, more or less laborious, occupations.

Pulsating Empyema.

728. Empyema, forcing its way through the costal pleura, may form one or more swellings under the skin, which rise and fall, if the parietal communication be free, with the acts of expiration and inspiration. This respiratory pulsation has long been known. But, some years since, Dr. M'Donnell showed, what had not been previously recognised, that a sub-cutaneous purulent collection of the kind may pulsate, synchronously with the heart, strongly and expansively, and hence simulate an aneurism. The absence of thrill and of abnormal vascular sound or murmur, the presence of the ordinary signs of empyema, and often the situation of the pulsatory prominence, will clear up the diagnosis. For fuller information, the valuable paper of Dr. M'Donnell may be consulted.*

729. But an empyema may become pulsatile under circumstances more singular than these, and still more likely to confuse the practitioner,—I mean where there is no perforation of the costal pleura, and no accumulation of pus under the skin. I have twice,† in cases presenting all the ordinary physical

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* Dublin Journ. of Med. Science, March, 1844.

† The first of these cases was seen (April 3, 1843) in consultation with Sir James Clark and Mr. Kingdon. Shortly after the first edition of this work was published, I had the gratification of receiving from Mr. A. Kempe of Exeter the particulars of a case observed by himself, completely according

signs of empyema on the left side, with displacement of the heart to the right, seen the inner part of the infra-clavicular and mammary regions close to the sternum, pulsate visibly, heavingly, and with sufficient force to jog the head at the end of the stethoscope,—the aorta, as proved by *post-mortem* examination, being of natural calibre. In both cases, while the side generally was greatly dilated, gentle local bulging was manifest in the site of the pulsation. The circumstances that determined the diagnosis in favour of pulsating empyema, were the absence of vascular murmur at the seat of pulsation; the fact that the two sounds heard at this spot were very weak, and gradually increased in intensity, as the stethoscope was carried towards the heart; the absence of thrill below and above the clavicles, and of undue impulse in the latter situation; the perfect equality of the radial pulses; and the total absence of signs of concentric pressure, venous, trachæal, or œsophageal. Taken singly, no one of these characters could be held conclusive, but the entire series formed a most serious body of evidence against the admission of aneurism. In both cases, the pulsation disappeared with the absorption of the fluid, and the return of the heart to its natural position: death arose from independent causes. In these cases the pulsatile character was already present when I first saw them: the diagnosis would be easier, if impulsive action of the kind made its appearance in a case of empyema while under observation; the sudden occurrence and great amount of the phenomenon, unattended with any of the ordinary evidences of aneurism, would guard the physician against error.

Pulsation conveyed to empyema, and simulating aneurism by its force and heaving character, seems to be merely an excess of that slight fluctuation movement in the fluid which is not very uncommon in ordinary cases.

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with the description in the text. In Mr. Kempe's very interesting case the pulsation was perceptible even at the upper two-thirds of the left back, as well as below the clavicle.

Pleurisy connected with Mammary Cancer.

730. Among the most frequent secondary morbid states, in cases of cancer of the female breast, stands pleurisy,—with or without cancerous patches under the pleura. Where subpleural cancer is not present to act as a direct irritant of the serous sac, it is probable the subinflammatory action, on the confines of the diseased gland, travels through the intervening tissues to that membrane. The frequency with which, in cases of extirpation of the diseased breast, pleurisy on the implicated side proves the immediate cause of death lends support to this view.

HYDROTHORAX.

731. Hydrothorax, dropsy of the pleura or accumulation of serous fluid without inflammation-products, occurs actively, passively, or mechanically. Of the former kind is the true hydrothorax, occasionally putting the close to existence in cancer of the mamma, and also occurring, instead of actual pleurisy, in some cases of Bright's disease: on the whole, this variety is very rare. In the great majority of cases, hydrothorax is passive or mechanical, occurs as a part of general dropsy, or is produced by obstructed circulation through the lungs and heart,—especially the right side and tricuspid orifice.

732. In hydrothorax the pain and "stitch" of pleurisy are wanting, and there is no tenderness under pressure. There is less cough, and may actually be none. But the mechanical effects of hydrothorax are commonly more serious than those of pleuritic effusion, for the simple reasons, that hydrothorax is generally double, and sequential to more or less serious organic disease, already disturbing the respiration and circulation,—pleurisy, on the contrary, generally single and primary. Hence the dyspnœa may be excessive, with constant orthopnœa, and extreme lividity of the face, anxious countenance, clammy

perspirations, and coolness of the expired air. If the patient can lie down, he does so on the back, with slight inclination now to one side, now to the other.

733. The physical signs agree in some, disagree in other points, with those of pleurisy. Dulness under percussion, moveable in area, with the changed posture of the patient; distant, weak, or suppressed respiration; bronchophony, occasionally ægophony or deficiency of resonance; total loss of vocal fremitus, and occasionally peripheric fluctuation, are common to the two affections. But in hydrothorax there is no friction-sound or fremitus, and both sides are commonly affected, while in pleurisy one only generally suffers. Dilatation of the side and flattening of the intercostal spaces may exist in both diseases, but relatively reach a higher point in the inflammation than in the dropsy. The heart and mediastinum are but little displaced sideways in hydrothorax, because the disease is generally double; the diaphragm may be very considerably depressed.

734. If the dropsy be of the irritative kind, moderate cupping is beneficial, unless some distinct contra-indication exist; under all circumstances, dry cupping is advisable. If there be no marked œdema of the walls, the chest should be blistered. Painting the surface with caustic iodine, or ioduretted frictions, sometimes obviously promote absorption. The internal remedies are diuretics and purgatives.

Inasmuch as hydrothorax is commonly either a local manifestation of a diathetic disease, or the effect of irremediable mechanical mischief in the heart, little, as a rule, can be expected from paracentesis,—at least in the way of permanent cure. But great temporary relief, and even prolongation of life, may be secured in urgent cases by the operation; and where asphyxia is threatened by double hydrothorax, it appears to me that puncture should at once be had recourse to.

PNEUMOTHORAX AND HYDROPNEUMOTHORAX.

735. By pneumothorax is understood accumulation of gaseous fluid in the pleura: the various modes of its production, real and alleged, may be classified as follows:

I. *No communication existing between pleura and external air.*
  (1) Gangrene of pleura; (2) Chemical decomposition of pleural fluid; (3) Air replacing sero-purulent fluid, suddenly absorbed; (4) Secretion by pleura.

II. *Communication between pleura and alimentary canal.*
  (5) Rupture of œsophagus into pleura.

III. *Communication with atmosphere through opening in chest-wall.*
  (6) Penetrating wounds of thorax.

IV. *Communication between pleural sac and bronchi.*
  a. Traumatic:—(7) Tearing of lung-surface by broken ribs; (8) Violent contusion of chest, tearing lung without costal fracture.
  b. Perforation, from disease opening the pulmonary pleura from within outwards, or centrifugal:—(9) Tubercle; (10) Gangrene; (11) Diffuse Pulmonary apoplexy; (12) Hydatids; (13) Cancer; (14) Emphysema; (15) Abscess; (16) Rupture in pertussis; (17) Excavated bronchial glands opening into the pleura and bronchi.
  c. Perforation, from disease opening the pulmonary pleura from without inwards, or centripetal:—(18) Empyema; (19) Parietal abscess.

736. As concerns Class I.: Gangrene of the pleura and decomposition of solid or fluid materials within its cavity, act in excessively rare instances, as the causes of non-perforative pneumothorax. It seems admissible, too, as a bare possibility, that, in those very exceptional cases of pneumonia, where true tympanitic resonance exists directly over the hepatised parts, that resonance may be caused by air secreted by the pleura [141-143]. Of any other kinds of simple pneumothorax I know nothing clinically; nor have I ever seen a case justifying belief in the real existence of general idiopathic pneumothorax.*

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* It has been too much the habit to pronounce pneumothorax idiopathic, whenever a superficial examination of the surface of the lung fails to detect an opening in the pleura. A crease in the substance of the organ, or a patch of exudation-matter will effectually conceal even a good-sized opening from view.

It has been taught on the evidence of a case recorded by Itard, that absorption of purulent matter may directly entail exhalation of air by the serous membrane. An old sufferer from empyema died with little pus and much gas (the latter, judging from the signs, of about three weeks' duration) in the pleura. The gas was of excessive fœtor, not however, it is held, from primary decomposition of the pus, but from chemical changes induced in healthy pus by gas originally odorless: this explanation is purely hypothetical.

737. Examples of Class II. are infinitely rare; and Class III. belongs altogether to surgical practice. Pneumothorax, depending on internal disease, is in the vast majority of cases produced by perforative diseases of the lung, falling under Class IV.,—and probably in 90 per cent. of instances of pulmonary perforation the affection causing it is tuberculous. It is true that of 147 recorded cases of pneumothorax, collected by M. Saussier, eighty-one only were phthisical. But tuberculous perforation is an every-day affair, which passes unnoticed; perforations from gangrene, vesicular emphysema, hydatids, pulmonary apoplexy, abscess, and other very rare causes, are greedily caught hold of, and registered. The number of the latter published, consequently, gradually swells out of proportion with their real frequency.

738. Of eighty-four cases of tuberculous perforation, seventy-four collected from various authors, ten observed by myself, fifty-five were examples of the occurrence on the left, twenty-nine on the right, side. The pleura commonly gives way postero-laterally in the area comprised between the third and sixth ribs. Perforation may occur at all periods of tuberculous disease,—and often depends on fresh deposition directly beneath the serous membrane: M. Louis records a case, where the

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The only trustworthy test is afforded by insufflation of the lung from the main bronchus under water. And supposing this test fails, it simply proves the absence of existing free perforation; such perforation may have existed, and have been closed up completely in a few days.

membrane gave way in a fortnight after the first appearance of phthisical symptoms.

739. The quantity of gas and fluid varies greatly; it may be immense. In the case, frequently referred to further on, 119 cubic inches of gas and 214 cubic inches of sero-pus were collected; the amount of the former was sufficient to float the body in a bath, with the affected half of the thorax raised out of the water.

740. In perforative hydropneumothorax the composition of the gas accords very closely with that of expired air. How comes this, seeing that the air enters the pleural sac in inspiration? The serous membrane, it has been suggested, acts, as lung would, on the air entering its cavity,—a most palpable error. In point of fact the pleural air though, physically speaking, inspired, is, chemically speaking, expired, inasmuch as it has passed through the lung. Besides, in virtue of the law of diffusion of gases, the gas in the pleura must be of the same composition as that in the adjoining lung.

741. From what has been said on the rarity of various forms of pneumothorax it follows that, in actual practice, the invasion-symptoms of pneumothorax, are equivalent, provided we exclude traumatic cases, to those of perforation of the pulmonary pleura. And these symptoms are, first, sudden sharp pain in the side, often of agonising, overwhelming severity; secondly, a sensation of rupture of tissue within the chest, followed by a feeling as if fluid trickled or poured down the side; and, thirdly, intense dyspnœa. If these three symptoms suddenly and unmistakeably appear in a phthisical person, the diagnosis of perforation would rarely be at fault; but the second is often wanting; and I have known perforation occur, as proved by physical signs and inspection after death, without any one of the three announcing its occurrence. The physical signs are the only unfailing evidence. After a time, the sensation of dyspnœa may wear off; I have found a man's respira-

tions fifty-two in the minute without his feeling any of the sufferings of difficult breathing.*

742. Air effused into the pleura acts as an irritant on the pleural surface; fluid is produced, and the compound state of hydropneumothorax established. Fluid is often demonstrably present within twenty-four hours after perforation; and probably begins to be formed almost immediately. The patient lies in various postures, but most commonly and steadily on the back, inclining to the sound side, with the head more or less raised; orthopnœa also occasionally exists at the moment of, and for a variable time, after perforation: all this may change, when effusion of fluid takes place. The pulse is excessively frequent; but the respiration relatively more so: I have known the ratio perverted into one of 2·3 to 1. The countenance is pinched, anxious, and imploring; the lips, cheeks, and face generally more or less livid; the sleep consists of fitful dozes at rare intervals; the skin is moist, sometimes bathed in cool clammy perspiration. The voice habitually loses strength greatly, and almost complete aphonia has sometimes been observed. Œdema of the affected side of the thorax is, at the least, very rare, before pneumothorax has been accompanied, and this for some time, with pleuritic effusion.

743. The prognosis of perforative pneumothorax is very difficult to establish at the time of its occurrence. It may prove fatal within twelve hours, a few days, or a week or two,—or the patient may completely recover from its immediate effects. The clue to these differences is by no means easily found; for,

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* Plympton, Clin. Lect. loc. cit., p. 575. I well remember, too, an ex-dragoon, originally of athletic form, in the third stage of phthisis, as an out-patient of the Consumption Hospital, coming to me one day with somewhat of a more axious facies than usual, and complaining of a slight pain below the right nipple of some hours' duration. He had walked without difficulty upwards of a mile to the hospital, and yet the right chest was so full of air as to have pushed the heart to the left axilla: all the signs of perforative pneumothorax were perfectly developed, and death occurred within a fortnight from the consequent pleurisy.

although in some cases the slight amount of previous pleural adhesions (a state permitting great accumulation of air) and considerable advancement, locally and constitutionally, of the disease causing perforation, appear to account plausibly for a rapidly fatal issue, death has been of tardy occurrence in some cases of the sort, and rapidly ensued, where the attendant circumstances were precisely the converse. Nor does an examination of the state of the non-perforated lung aid very materially in settling the question; for, if, while in the main tolerably healthy, peripheral tubercles even to a small amount are lodged immediately beneath the pleura, that membrane may give way at any moment, and immediate death ensue; whereas experience proves that in some cases of prolongation of life after the perforation of one lung, the other has been the seat of numerous deep-seated cavities.

When the occurrence is not fatal at once, the perforation may either undergo closure by lymph, or remain pervious. I have seen two cases of the former kind, in which all signs of air and fluid in the pleura had disappeared in the course of two months after the perforation: here the cure was absolute. Even when the opening remains pervious, the compound disease is not necessarily fatal. Laennec refers to a phthisical case where the signs of fistulous hydropneumothorax continued at the end of six years; and instances have now been collected in some numbers in this country and abroad, positively proving that not only may life be prolonged, but excellent health enjoyed, while succussion-sound is well audible in the side: still such cases are completely exceptional.

744. The physical signs of pneumothorax are very significant. The chest-motions suffer more or less extensively in freedom; they may be absolutely null at the lower part of the affected side: where there is any play, the intercostal spaces deepen during inspiration greatly. The vocal fremitus is weakened or annulled; the width of the side increased to the eye and to measure; the interspaces widened, and even bulged outwards,

inferiorly, may be natural superiorly. The percussion-sound, increased in clearness, acquires tympanitic quality, retaining this until the accumulation of air becomes excessive [140]. Local pneumothorax, at least in front of the trachea and large bronchi, may give an amphoric note. If the quantity of air be moderate, the respiration is of distant, weak type,—if considerable, absolutely suppressed. The conditions of vocal resonance vary; there may be mere nullity of sound; in some cases, the resonance is loud and diffused; and possibly it may be sometimes accompanied with metallic echo. The heart's sounds are, as a rule, obscurely transmitted through the air in the chest. The mediastinum, heart, and diaphragm are more or less displaced, and the clearness of percussion may extend considerably beyond the middle line.

745. In cases of simple hydro-pneumothorax the signs are a combination of those of pleuritic effusion and of pneumothorax,—the former at the lower, the latter at the upper, part of the side.

746. When hydropneumothorax coexists, as is the rule, with perforation of the lung, fluctuation may be felt both by patient and observer, when the chest is abruptly shaken; peripheric fluctuation exists to its maximum amount;* while Hippocratic succession-sound, with its metallic ringing splash, readily produced one day, may be absent the next: these three signs may exist, although closure of the perforation has taken place. The dull sound of fluid, and the tympanitic resonance of air, are found, according to the posture of patients, the former inferiorly, the latter superiorly: but the exact sites of both may be variously changed (unless adhesion, which is rare in well marked cases, interfere) by altering that posture. Between the areæ of tympanitic air-resonance and the dull sound of the fluid a narrow space may exist furnishing an amphoric note.† The respiration is amphoric, with or without metallic echo or

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* Plimpton's case, loc. cit.

† Ibid.

tinkling; and the cough and vocal resonance are similarly echoed: the amphoric quality is most obvious, the nearer the perforation auscultation is performed. The phenomena of displacement of organs are carried to the highest possible point. The heart's sounds are commonly weakened in their passage across the distended pleura; but they are sometimes echoed within it. The respiration-sounds in some cases, when the fistula is entirely open, amphoric at one time, are simply weak and distant or actually suppressed at another; under these circumstances a peculiar inspiratory sibilus is sometimes heard all over the side, and probably depends on escape of air through the chink in the lung.* The chosen posture may be on the diseased side; but the same patient will change his mode of lying frequently. Orthopnœa is not common.

747. The treatment of perforative tuberculous pneumothorax is palliative. In cases of severe suffering, if the patient be seen immediately, or shortly after, the pleura has given way, while his agony, both of pain and dyspnœa, exists in all the intenseness of novelty, bleeding suggests itself as a means of relief. The quantity of oxygenating surface has been suddenly reduced, and the sudden disparity between that surface and the mass of the blood might, or would, I think, be somewhat lessened by diminishing the quantity of the latter. Whether this be the explanation or not, venesection does give very notable relief, and renders subsequent inflammation of the pleura less violent. It should then be had recourse to, where the patient's strength has not been materially impaired by the previous disease. In doubtful cases, moderate cupping of the side may be substituted,—or if the effects of even this mode of depletion be feared, general dry cupping of the chest. It is singular what relief, both of pain and dyspnœa, is sometimes afforded by the latter process. Repeated flying blisters to the side are also most valuable agents. The bowels must be kept moderately

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* Plimpton's case, loc. cit.

open, perspirable action of the skin promoted, the strength maintained by nutritious animal jellies and broths, or by meat, if the patient's digestive powers be not enfeebled: while inflammatory symptoms are controlled, as they arise.

Various anti-spasmodics are useful in mitigating the dyspnœa, —lobelia inflata, cannabis indica, belladonna, stramonium, aconite, and above all, opium. I have seen musk, in doses of five grains, afford great relief.

748. In tuberculous pneumothorax, paracentesis can only be regarded as palliative, and, what is worse, temporarily palliative. Still, as the operation does not obviously place the patient in any way in a worse position than he had been before, and as it often gives new existence for awhile, there can be no objection to its employment, and to its repetition, when physical signs show that the mediastinum and the non-affected lung are seriously encroached upon.

It is difficult to lay down a rule for other varieties of perforative pneumothorax; recorded cases are deficient in detail for our guidance, and to me it appears that where recovery has ensued, it would have occurred without the operation. If pneumothorax were suddenly produced in a fit of violent coughing, as in hooping-cough, and the patient had previously exhibited no evidence of organic disease of the lung, I should hold it advisable, especially if the symptoms were urgent, to puncture the thorax.

SECTION V.—THE PULMONARY PARENCHYMA.

NEUROSES.

749. The pulmonary tissue is highly endowed with a special sensibility, as proved by the exquisitely painful sensation produced by want of air. Here is one of the chief sources of suffering in all forms of dyspnœa.

750. On the other hand the parenchyma of the lung is, not only, under ordinary circumstances, feebly provided with

common sensibility, but even in the state of inflammation is very rarely the seat of pain, at least of pain demonstrably referrible to itself. Still, pain has occasionally been noticed in central pneumonia, where the pleura had wholly escaped; and various anomalous, and more or less painful, sensations felt by phthisical patients, deeply within the chest, apparently originate within the pulmonary texture. Certain morbid processes in the parenchyma appear then to irritate the branches of the pulmonary nerves.

751. The only physical condition I have ever succeeded in connecting with these painful sensations, and this only in cases of phthisis, is jerking rhythm of the respiration. The quality of the breathing-sounds also may probably be roughened by their existence.

752. These sensations are either greatly relieved or altogether removed by counter-irritation, and emollient and anodyne inhalation. They are not so directly, as might be expected, modified by anodynes taken internally.

ACUTE PNEUMONIA.

753. Acute inflammation of the pulmonary tissue, or pneumonia, is habitually divided into three anatomical stages,—those of engorgement, of red hepatisation, and of gray, or suppurative, hepatisation.

754. In the stage of engorgement, the external surface of the affected parts is of livid or violet colour, or mottled with both tints and with dull red; the weight of the organ is increased,* its elasticity diminished; it retains marks of pressure like an œdematous lung; and crepitates imperfectly. On section reddish spumous serosity escapes in greater or less quantity;

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* The weight assigned to healthy lungs in books of anatomy is decidedly above the reality, in consequence of the measurements being habitually made from organs more or less congested *in articulo mortis.* Lungs really normal in all respects weigh scarcely ten ounces. Tuck, U. C. H., Females, vol. ix. p. 67, ætatis 52,—the right lung weighed nine ounces.

the spongy character of lung is still to be recognised; the consistence is diminished. The permeability to air is lessened, not destroyed; the tissue floats in water, even after forcible pressure with the fingers.

755. In the second stage the section of the inflamed part presents a rude resemblance to the liver, hence the name of red hepatisation. The outer surface is commonly redder, less livid, than previously; the specific gravity and absolute weight are more or less increased, the tissue rapidly sinking in water; all natural elasticity is gone; the texture quasi-solid. On section the surface proves generally of red tone, not uniformly red, but mottled, or, as it were, veined; a variegated character increased by the different hues of the bronchi, vessels, black pulmonary matter and cellular septa. Fluid escapes on pressure, but in less quantity than before; it is thicker, more bloody, and more or less completely airless. The surface looks smooth, planiform, or beset with a multitude of red, granular-looking eminences, particularly obvious if the texture be torn, and evidently depending on repletion of the air-vesicles with exudation. The absolute volume of the organ increases; hence it encroaches on the mediastinum, enlarges the side and takes the impress of the ribs. Its weight may increase to six or eight times the natural amount.

In new-born infants the granular aspect is quite wanting, even when the tissue is torn; in advanced age granulation on a large scale, with empty spaces interspersed through the lung, occasionally presents itself.

756. There is a variety of this stage in which the inflamed tissue of vinous red, or livid tint, heavy, impermeable, containing very little fluid and no air, with a uniform smooth non-granular section, proves really firm and resistant; this state is known as carnification. I have only seen it where the lung appeared, previous to inflammation, to have undergone condensation by pressure.

757. The inflamed lung is sometimes peculiarly soft, almost

pulpy, breaking down under the slightest pressure, and gorged with thickish claret-coloured non-aërated fluid ;—so-called splenisation. Its peculiarities seem to depend on the state of the blood ; it is most common in persons worn down by previous old-standing disease.

758. In the stage of interstitial suppuration several of the properties of red hepatisation remain: dense, heavy, enlarged, impermeable, sinking in water, and of even greater friability, the tissue is however more or less completely changed in colour. The general tinge becomes yellowish or yellowish-gray,—sometimes granite-like from the black matter of the lung.

759. Dr. Stokes contends that a stage of pneumonia prior to that of engorgement exists,—distinguished by dryness and bright vermilion colour, and constituted by intense arterial injection. Skoda denies this, and with Rokitansky looks on the colour in question as the product of anæmia.

My opinion coincides with that of Dr. Stokes. The vivid arterial tint in question is seen to perfection in rapidly fatal cases of acute miliary tuberculisation, in persons presenting none of the acknowledged characters of anæmia, but many of those of pneumonic irritation. Besides it may exist in one lung and be absent in the other. (Knowles, U. C. H., vol. x., p. 175.)

760. During the stages of arterial injection and engorgement, the blood-vessels are alone implicated. In that of red hepatisation the capillaries yield their contents,—plasma, water and red disks,—to the tissue around. Cell-formation occurs both within and between the air-vesicles; fibrinous casts of the vesicles and ultimate tubes are also discoverable. In the third stage true pus-cells abound in some cases ; are rare in others ; and in yet others cannot be found, exudation-cells being alone visible. The naked-eye appearances of suppuration are consequently sometimes simulated by liquified exudation-matter.

761. Abscesses, varying in size from that of a nut to that of the clenched hand, one or more in number, sometimes form in a hepatised part ; commonly seated near the pleura, they may

perforate this membrane and produce pneumothorax. The cavity of such abscesses is irregular, traversed by bronchi, vessels and fragments of tissue; the walls are formed of pulmonary substance, either bare, or lined with a pyogenic membrane, which sometimes forms with great rapidity, certainly in the course of six days. Minute sloughs may form secondarily on the walls. The pyogenic membrane may become pseudo-mucous, and the cavity fall into a quiescent state symptomatically; or actual cicatrisation may occur.

Amid the contents of pulmonary abscess, essentially composed either of laudable or sanious pus, fragments of parenchyma are occasionally discernible.

Abscess is more common towards the apex of the lung than elsewhere; advanced age seems favourable to its occurrence.

762. The inter-lobular and inter-lobar cellular-tissue may inflame and suppurate;* or, as shown by Dr. Stokes,† the pleura may be dissected from the lung by suppuration.

Physical Signs.

763. The physical signs of acute pneumonia vary with the amount of consolidation, and the nature of the secretion into the cells,—hence with the various stages of the disease.

764. Are there any special signs of the stage of arterial injection? It would be difficult to prove the affirmative positively: but previously to the occurrence of crepitant rhonchus, it is certain the following conditions may sometimes be detected. The breathing-sounds reach the ear harsher, rougher, and of higher pitch than natural from the affected part, provided this be near the surface; if the affected structure be deep-seated, on the contrary, exaggerated respiration, from the intervening healthy tissue, is heard: the percussion-sound is unchanged. I have now seen a fair number of cases in which such exaggerated

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* Carswell's Drawings, U. C. Museum, No. 57, C. b. 573.

† Dublin Journal, 1833.

respiration, coupled with febrile excitement, and slight pain in the side, were the earliest indications of a central pneumonia, eventually travelling to the surface. But this is not always so: M. Grisolle holds that "in the great majority of cases, if not in all, weakness of respiration, often attended with loss of purity and of softness," marks the outset of the disease. The same writer states that he has, in a large proportion of cases, found weak respiration, in the neighbourhood of already hepatised lung, the precursor of signs of consolidation: this latter statement I can confirm; in some such instances no crepitation can be caught from day to day, while the spread of the disease is indicated by percussion-dulness and bronchial breathing.

It would appear, then, a disturbed state of respiration, not always of the same type, and hence without precise signification, is the only condition referrible to this stage.

765. In the engorgement-stage, the movements of expansion and of elevation are, if pleuritic pain be present, somewhat restrained: the vocal fremitus maintains its natural standard; the percussion-sound is more or less dull, according to the amount of engorgement; the respiratory murmurs are weak, suppressed, or masked by rhonchus in the affected parts and their immediate confines, exaggerated in those at some distance from them and in the opposite lung; the vocal resonance is somewhat intensified, and slightly sniffling in quality.

In the great majority of cases, watched from the outset, crepitant rhonchus is more or less extensively heard during this stage; but, in hospital practice especially, the period of its existence has often passed when the patient is first seen. There are instances, too, in which, though the pneumonia be developed under observation, no crepitation occurs: here the intra-parenchymatous exudation seems to be thrown out so rapidly, as to arrest all breathing action in the actually inflamed part, *ab initio*. And in a word, the diagnosis of pneumonia must often be made without the help of true crepitant rhonchus.

766. Whether in the stage of red hepatisation, pneumonia, unattended with pleurisy, produces general expansion of the side, has been a theme of constant dispute. M. Woillez, maintaining the negative, justifies his opinion by a reference to the physical relations of the lung to its containing cavity, corroborated by the results of direct mensuration in two cases; in neither of these instances was the least degree of expansion detected. M. Grisolle obtained similar results from circular and antero-posterior admeasurement in four cases; nevertheless, he believes, upon the evidence of two others, that the inflamed lung may, quite independently of pleuritic effusion, determine "general or partial dilatation." In one of these, where the disease occupied the upper lobe, and especially its anterior part, slight bulging of the infra-clavicular region was detected on the patient's admission, the third day of the affection. This bulging having gradually increased with the progress of hepatisation, M. Grisolle considers himself warranted in referring its appearance to the inflammation of the lung; the *post-mortem* examination proved the absence of pleuritic effusion. In the other instance, bulging of the infra- and post-clavicular regions was observed to subside gradually with the resolution of the disease. Now, neither of these cases proves the fact of general expansion, as admitted by M. Grisolle; though there does not appear to be any plausible objection to them as demonstrating the occurrence of partial expansion. But in a small minority of cases, I have myself found positive, though slight, increase of width at the base of the affected side. Besides, the evidence of slight encroachment of the inflamed organ on the mediastinum may very frequently be obtained by percussion at mid-sternum; the attendant dulness may reach a quarter of an inch beyond the middle line.

The expansile movement of the chest, and even the abdominal movement, are lessened in amount on the affected, somewhat augmented on the sound, side,—and this in simple pneumonia with extensive consolidation, quite independently of the influence of severe pain. But the movement of elevation is not by any means so much obstructed, according to my observation, as that of expansion; a circumstance which will perhaps account for the contradictory opinions held on the subject. In certain cases of pneumonia with a slight amount of plastic exudation on the pleural surfaces, friction phenomena are not to be discovered. To what can this be attributed, but to the diminished expansion of the lung, and deficient mobility of the thoracic walls?—while, on the other hand, it would be preposterous to explain the diminished motion by the interference of the painless pleuritic exudation supposed.* The respiratory play is impaired, not only by want of expansile, but of contractile power on the part of the lung: I have known the latter even more deficient than the former: the elasticity of the texture is wholly destroyed.

As a rule the vocal vibration is increased in intensity; sometimes simply maintained at the normal average;† while in cases of exceedingly extensive, and, as it were, massive hepatisation, it may actually fall below the natural standard. Fortunately this is rare. A vibratile tremor is sometimes conveyed to the surface through the lung from the heart.

The percussion-sound, habitually diminished in clearness to absolute dulness, with greatly increased parietal resistance, may under certain circumstances of locality of the inflammation, as originally shown by Dr. Hudson, acquire a tubular or even an amphoric quality; this, it is conceived, will occur if the solid tissue lie over a distended stomach, or between the large bronchi and the surface. Resonance of this quality may, however, occur at the posterior base at the right,* as well as on the left, side ; the respiration has then in the same spot more or less hollow character. In much rarer cases, the percussion-sound may be purely tympanitic over the hepatised lung. I have found this at the anterior base on the right side. [142.]

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* Even where the consolidation of the bases is extreme, and the amount of pleural lymph very small, there may on the other hand be well marked friction-sound. Skinner, U. C. H., Males, vol. ix., p. 98: here the lungs, though emphysematous, weighed 37½ oz. and 42½ oz.

† When pneumonia supervenes on extensive emphysema, this will be the case; the vocal resonance may be less marked over the pneumonic than the simply emphysematous parts, and yet the fremitus be well maintained over the former on the right side. Skinner, U. C. H., Males, vol. ix., p. 95.

The respiration is blowing, of the diffused or tubular varieties, sniffling, metallic, abrupt, and divided in rhythm ; exaggerated in distant parts ; no crepitant rhonchus is produced in the actually hepatised part, but may, or may not, be heard on its confines. Bronchophony, sniffling, muffled and intense, sometimes quasi-œgophonic, bronchial cough, and unnatural intensity of transmission of the heart's sounds, complete the list of discoverable signs. Of all these, the intensified vocal fremitus, the metallic tubular respiration, and the percussion-dulness are the most important.

Sonorous rhonchus was regarded by Laennec as an occasional effect of compression of the bronchi by the indurated pulmonary tissue. However caused, such rhonchus is very uncommon in pneumonia, and is probably, as is generally held, the result of bronchitis. I do not remember to have observed it, to any notable amount, except in the pneumonia of infants, in whom, as is fully ascertained, the co-existence of these two inflammations, or the transition of bronchitis into pneumonia, are much more common than in the grown person. Were Laennec's opinion respecting the mode of production of dry bronchial rhonchus in pneumonia correct, its occurrence would necessarily be very frequent, instead of being singularly uncommon. Possibly, the co-existence of dilatation of the bronchi may have some indirect influence, in the production of sonorous rhonchus in pneumonia ; at least, it is certain that the period of life at which the rhonchus is most common—namely, infancy—is precisely that at which dilatation of the bronchi most commonly attends hepatisation,—as shown by Dr. Ogier Ward.*

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* Lewis, U. C. H., Females, vol. ix., p. 320.

767. In certain rare cases of pure red hepatisation, the physical signs, with the exception of dulness under percussion, are wholly wanting; the vocal fremitus is deficient; there is neither vocal resonance nor respiration heard on the affected side. These peculiar conditions seem probably to depend on such extensive closure of bronchial tubes, as to prevent the possibility of reinforcement of respiration or of vocal sound within them, while the side is motionless, and the powers of conduction of the lung (which, we know, vary widely according to the precise acoustic conditions of the hepatised substance [261]) are annulled. Pressure on a main bronchus by an enlarged gland or tumour, if of sufficient size to obstruct the tube completely, will produce this effect on the signs; but such pressure is not a necessary condition.

A more singular state of things still may exist. Tubular respiration may be intermittent,—sometimes existing, sometimes no respiration being audible. I have traced this in one instance to pressure on the main bronchus of the affected side,—obviously acting at various moments with different amounts of force.†

768. In the stage of gray hepatisation, interstitial suppuration, or liquefied exudation-matter, the signs are essentially the same as in the previous stage. If suppurating points freely communicate with bronchial tubes, loose liquid rhonchus, more or less thin and high-pitched, according to the more or less completely purulent quality of the fluid in the lung, will be produced. But such communication is, of course, a matter of accident; and, admitting the occurrence both of communication and rhonchus, I know of no character by which such rhonchus can be positively distinguished from bubbling rhonchi arising in tubes surrounded by consolidated tissue.

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* Medical Gazette, 1838.

† Beckett, U. C. H., Males, vol. v., pp. 231, 274.

There may, too, as in the previous stage, and through the same causes, be complete absence of all auscultatory signs. I saw a remarkable case of this kind in 1847, with Dr. Storrar: there were neither respiration-sounds nor rhonchi audible in front of the chest, directly over lung in a state of yellow semi-liquid infiltration; dulness, the most absolute, was the only physical sign in the spot.

769. The *resolution* of pneumonia may occur, in some very rare instances, before red hepatisation has been effected; but in the vast majority of instances, the disease has advanced to the hepatised stage before resolution commences. The signs of this favourable event are, first, a change in the quality of the tubular breathing; it becomes less sharp and metallic, more open,—in a word, of diffused blowing type, which serves as transition to a harsh and weak respiration; the bronchophony rapidly loses its peculiar sniffling quality, but holds on to a slight amount for some time; while either redux crepitant or fine bubbling rhonchus, becomes, and remains, audible for a variable period. The percussion-sound gradually grows clear, and, as a rule, in cases of favourable course, more rapidly recovers its natural character than after the absorption of pleuritic effusion. I have known the tone scarcely different from that of health in a spot where, three days before, there had been the most perfect dulness: but such rapid resolution is, unfortunately, very rare. It has been made a matter of much dispute, whether retraction of the chest walls may occur in the advanced periods of pneumonia, where no suspicion can exist of the presence of pleuritic effusion. Dr. Stokes teaches the affirmative. Contraction may be observed, he states, in cases where the lung has been long indurated and still continues impervious, and may even co-exist with gradual and ultimately perfect resolution. In all cases where he has observed this contraction, the primary disease has been of the *typhoid* type; in one instance of the kind, the contraction seemed to affect the whole side more than is general in pleurisy; in other cases it

was very similar to that of empyema. M. Woillez maintains, on the contrary, that where contraction occurs in pneumonia, there has always been some effusion into the pleura—in fact, pleuro-pneumonia,—and that the process of contraction is the same as in simple pleurisy. M. Grisolle adopts the same views respecting this question as M. Woillez. In nine cases of *simple* pneumonia, antero-posterior and circular admeasurement failed to detect any diminution of size during the progress of convalescence.

Some years since I observed a case of extensive pneumonia of the left side, in which indisputable depression of the *latero-anterior* part of the chest gradually took place during the progress of recovery. As far as physical and symptomatic evidence can decide the point, there was certainly neither notable pleural exudation nor liquid effusion in this instance; though, as in Dr. Stokes's cases, there was no *post-mortem* examination to decide the question, I cannot help feeling persuaded that simple pneumonia may entail the alteration of shape which I thus believe I have actually witnessed. Perhaps, all things considered, the most efficient agent in producing depression of the chest after pleurisy, is the contraction of the plastic matter exuded on the pleural surfaces. Why should not the same contraction, occurring as a law of its existence, of exudation poured into the substance of the lung, cause similar alteration in the form of the thorax? I say similar, not the same in amount, because, in the case of pleurisy, there is another well-known cause of depression, which does not exist in the instance of pneumonia. It appears curious that M. Grisolle, who professes to have seen the size of the lung, enlarged by interstitial exudation solely, gradually return to its natural state, should maintain depression of the surface to be impossible. What is to prevent the tendency to diminution of bulk from gradually bringing the lung to a less volume than in health; and this once effected, will not depression of the parietes inevitably follow?

770. The rhonchus crepitans redux, like the true crepitant, suggests the idea of crepitation; but the crepiti constituting it are moister and commonly more suggestive of "bubbling;" they convey the impression of larger size; are more slowly evolved, rarely, if ever, occurring in abrupt puffs; are comparatively few in number; are more or less dissimilar to each other; somewhat irregular in occurrence; and frequently audible in expiration as well as in inspiration, though more specially appertaining to the latter.

Such are the ordinary characters of the rhonchus coexisting with the resolution of pneumonia. But in a certain number of cases its properties are much more similar to those of true crepitation: it possesses the same dryness, the same minuteness (I have never, however, observed redux crepitation of greater delicacy than the primary, as M. Grisolle appears to have done), and coexists with inspiration only. The observation of these facts, and of the vague manner in which the phrase "redux crepitation" is applied to the rhonchi existing in lungs undergoing the resolution of pneumonia, coupled with examinations made for the express elucidation of the point, has long led me to the conclusion that under that phrase are confounded two very different phenomena. These phenomena are a fine bubbling rhonchus, and a true returning primary crepitation. The former is by far the more common, has all the characters of a humid rhonchus, and is, I can scarcely entertain a doubt, produced in the minute bronchial tubes; the latter, which affects the characters of primary crepitation, is probably generated in the same seat and manner as this.*

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* It will be observed that the rarity of true redux crepitation is in accordance with the theory which localises the primary rhonchus in the parenchymatous exudation [205]: it is in truth unlikely that the physical condition of the interstitial plastic exudation should often be similar at the two opposite periods of the malady, and hence improbable that a given phenomenon depending for its existence on that condition should frequently occur with identical characters at both those periods. But the thing may, *à priori*, be conceived a possible occurrence; and so we find by observation, that the effect

The immediate cause of the fine bubbling rhonchus attending resolution is probably the passage of air through fluid contained in the minute bronchi: and this fluid may be the result either of capillary bronchitis, or be merely on its passage from the previously engorged and now œdematous lung. This latter opinion seems calculated to throw some light upon the cause of a circumstance with which auscultators are well acquainted; namely, that the rhonchus of resolution of pneumonia sometimes lasts but a few hours, and at other times persists for weeks.

771. When, instead of the infiltrating form of suppuration, abscess occurs, the physical signs are of course peculiar. The student should remember that in diagnosticating pulmonary abscess, the first point, if possible, to be ascertained is, whether the signs of pneumonia have existed in the organ, which is suspected to be the seat of purulent collection. Admitting this to be settled in the affirmative, the special signs of abscess will vary according as the pus has been more or less completely evacuated, or is still retained.

First, in the case of a *pulmonary abscess, of which the contents are more or less completely evacuated*, the diagnosis is grounded generally on the fact of the signs of excavation supervening upon those of pneumonia. The percussion-sound is either dull with marked parietal resistance, or of tubular, amphoric, or cracked-metal quality; the respiration is cavernous, or tubular, accompanied with large-sized, thin metallic, echoing rhonchus. The vocal resonance may be bronchophonic, pectoriloquous, or null.

Secondly, in the case of *an abscess with its contents retained*, in consequence of want of ready bronchial communication, the

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which would follow, did it occur—that is, the re-appearance of the true primary rhoncus—*is* occasionally met with. On the other hand, were the primary rhonchus produced by bubbling in the interior of the vesicles, there is no obvious reason why the return of true primary crepitation should not be an invariable sequence of resolution.

signs are, of necessity, extremely obscure. There are none, indeed, of an absolutely distinctive kind. The dulness is marked, the percussion-sound may be tubular, the respiration tubular, and the vocal resonance strongly bronchophonic. But all this might have existed before the formation of abscess.

772. Sphacelus of the lung,—one of the very rarest terminations of acute pneumonia,*—would be signified by the signs of a cavity; the expectoration growing, at the same time, profuse, and of peculiar fœtor.

773. An œdematous state of the parenchyma may remain long after the resolution of pneumonia. The signs of this state are elsewhere described.

Symptoms.

774. A certain proportion of adults, varying between a sixth and a third of the whole number attacked, suffer from *prodromata* in the shape of general febrile disturbance, of from one to four days' duration, after the action of the cause, and before any local symptom of the disease makes its appearance. In the majority of cases the invasion is sudden.

775. The *invasion* of the disease is marked by rigors, followed by pain in the side, short cough, oppressed breathing, and sometimes cephalalgia and vomiting. Rigors are very rarely deficient; in a great proportion of cases they form the phenomenon of invasion, with greater frequency, indeed, than in any affections, except ague, and perhaps puerperal fever. The respiration is often accelerated greatly out of proportion with the pulse, at the very outset,—before any local symptom or sign points to pneumonia.

776. Among the symptoms of the *actual disease* stands *pain*

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* The rarity with which gangrene of the lung originates in acute sthenic inflammation is now generally recognised. Of 305 cases of pneumonia analysed by M. Grisolle, not one terminated by gangrene; and of 70 cases in various journals perused by him, 5 only could be considered positive instances of this mode of termination of the acute disease.

in the side (29 only of 301 patients escaped it; and in 161 of 182 it appeared within the first twelve hours; Grisolle): pain, generally seated below the nipple on the affected side; rarely corresponding with the seat of the pneumonia; depending commonly on co-existing pleuritic irritation, rarely on intercostal neuralgia or neuritis, in infinitely rare cases seated apparently in the lung substance itself; in the last case, always slight; in the two former, variable in amount; and increased by cough and parietal pressure.

777. *Increased frequency of breathing*, to a variable amount, is a constant symptom; occurs within the first few hours; and raises the number of respirations to from 24 up to 80 per minute,—from 30 to 50 being the more usual extremes. When the respiration reaches 70 or 80, suffocation seems threatened, and speech is obstructed; whereas 30 or 40 respirations per minute may exist without the patient being conscious of particular dyspnœa. The frequency of breathing does not depend on any particular seat of the pneumonia, nor even, singularly enough, on its extent,—at least necessarily. I have known double pneumonia attended with a less number of respirations than inflammation of a limited portion of one lung. Although marked acceleration of breathing is an unfavourable sign, it is not, even to the highest degree, of fatal augury: recovery may take place where the respiration has reached 80 per minute. Now the circulation does not increase in frequency in the same proportion as the respiration,—hence the ratio of the two becomes more or less perverted. Thus, for 100 pulsations per minute, there may be 60 respirations (I have seen this)—numbers giving a ratio of 1·7 : 1, instead of 4·5 : 1, that of health. The same perversion exists in those exceptional cases of pneumonia in which the pulse maintains a low frequency throughout. This perverted pulse-respiration ratio may, as I have found in several instances, *prove the first sign of pneumonia, appearing before crepitation or rusty expectoration;* as, *per contra*, a return to, or towards, the healthy standard, *may*

announce resolution some days earlier than the rhonchus crepitans redux.[*]

Cough, in the great majority of cases, occurs within the first twelve hours,—moderate in amount, rarely paroxysmal, more severe in double than single pneumonia, and diminishing, occasionally even ceasing, towards the close in fatal cases. It is accompanied in the majority of instances with *expectoration* of very striking characters,—sanguinolent, or rusty in tint, viscid, semi-transparent, adhering to the vessel, but slightly aërated, passing through various shades of orange, apricot colour, faintly greenish, and lastly becoming white—then opaque and of bronchitic qualities. In some cases the sputa are diffluent, watery and of dull brown, or even faintly blackish hue,—resembling liquorice juice, or prune juice—conditions of evil augury. The red tint of the sputa in the mass of patients comes on during the first forty-eight hours, and after the fourth day its appearance becomes very unusual; once developed, it may last from one to fourteen days. In some rare instances, the sputa continue white throughout; and in yet rarer ones, especially where the pneumonia is central, or of the upper lobe, there is no expectoration. If the expectoration in pneumonia be actually more or less profusely bloody—if there be hæmoptysis, in short—the pneumonia is tuberculous, according to my experience. Microscopically the rusty pneumonic sputum is composed of muco-epithelium, blood-disks, exudation-cells, amorphous patches of exudation-matter, oil-globules and granular fat, sometimes in large quantity, and moulded protinaceous casts of the ultimate bronchial tubes, rarely of the air-vesicles,—casts studded more or less abundantly with exudation-cells. True pus-cells are very rare. Accidental ingredients, such as biliphæin, are met with in certain diathetic varieties of the disease. Sugar exists in sufficient quantity, sometimes at the height of the inflammation to be discoverable by Trommer's test: the interference with oxidising processes in the hepatised tissue accounts both for the oil and sugar in the sputa [781].*

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[*] Craddock, U. C. H., Males, Clin. Lect., loc. cit., p. 144. I have observed the same perversion, antecedent by twelve hours to any physical sign of pneumonia, in a case of excision of axillary cancer.

Sputa, having all the characters now described, are absolutely pathognomonic of pneumonia. The naked-eye characters, taken alone, are not so formally distinctive; at least in some cases of mechanical congestion of the lungs, especially from mitral disease, the expectoration may be somewhat viscid, faintly aërated and semi-transparent, and the blood-tint rusty.

The *expired air* is sensibly colder than natural, especially when the respiration is very frequent; and the quantity of *carbonic acid* expired is, according to the experiments of Nysten, diminished. It would also appear that accumulation of *fat* occurs to a great amount in the inflamed parenchyma:† doubtless, from the impermeability of the lung, products, which should be oxidised, fail to undergo that change. The well-known fact, that pneumonia does not cause such rapid emaciation as many other equally dangerous diseases, deserves attention in connection with this peculiarity. Sugar also would probably be found, if the hepatised tissue were examined immediately after death.

The peculiar acid (pneumic) of the lung-substance, discovered by Verdeil, appears to undergo increase in pneumonia. In a single hepatised lung of a male there was a very considerably greater quantity of the acid, than in both lungs of a perfectly healthy guillotined woman.‡

778. Among the *general symptoms*, the state of the *pulse* is important. It may reach 140 or 160 beats per minute,—and

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* The freer the use of amylaceous food, the easier is the detection of sugar in the excreta of pneumonia, as of all other diseases in which sugar persistently or temporarily impregnates the liquid secretions.

† The natural proportion of fat to parenchyma being 10 per 100, it may rise in pneumonia to 50 per 100, according to M. Guillot.

‡ Robin et Verdeil, Chimie Anatomique, t. ii., p. 460.

in the mass of serious and fatal cases, is of much greater frequency than in those of recovery, generally attaining its maximum frequency by the third or fourth day. I have, however, known pneumonia run its course, both in old persons and in young adults, with a pulse never exceeding 60; in these individuals, however, the healthy standard has, on recovery, proved still lower. Unless when very frequent, the pulse is habitually full and resisting: when feeble and small, this sometimes depends on embarrassed circulation, produced by *distension of the right cavities of the heart* with blood. Fulness of the jugular vein sometimes appears under such circumstances,—possibly, sometimes, from actual pressure on the vena cava by the hepatised lung: jugular pulsation occasionally occurs, and pulsation even of the dorsal veins of the hand has, in rare instances been seen [507, 509]. The *blood*, buffed and cupped, is hyperinotic in the highest degree,—the fibrine ranging from 6 to 13 per 1000. There is a peculiar tendency to the formation of solid resistent coagula in the right heart, and in the pulmonary arteries, an obvious source of embarrassed circulation and other dangers; but I must express my dissent from the opinion of M. Bouillaud, that their formation is an invariable attendant on hepatisation.

779. The temperature of the *skin* is raised; its feel often pungently, acridly, burningly hot,—not more so in the vicinity of the inflamed part than over the rest of the chest. Sweating, sometimes copious, may occur towards the decline of the disease; and sudamina may form in abundance. I have known the temperature under the tongue reach 106° Fah.

780. The *digestive organs* sympathise. The *tongue* varies in appearance, but does not exhibit the adynamic ("typhoid") character in sthenic cases; *thirst*, scarcely in the ratio of the fever; *anorexia;* occasionally vomiting; tendency to constipation, or sometimes, especially in fatal cases and towards their close, diarrhœa, more or less profuse; mark the implication of the alimentary canal. *Jaundice* occasionally occurs, on the

whole more frequently when the right lung is affected than the left, but with little more frequency when the disease is seated in the lower rather than the upper lobe of that lung.

781. The characters of the urine, of ready clinical discovery, may be set down as follows, from the analysis of a considerable number of cases. The specific gravity ranges between 1012 and 1030,—the maximum being observed at the height of the disease, the minimum at the outset and at the period of convalescence. Crystals of uric acid occasionally, urates in abundance, coincide with deep colour, strong urinous odour, and with high specific gravity. Muco-epithelium occurs indifferently at all periods. Convalescence is sometimes coeval with a copious appearance of crystalline triple phosphate, or of oxalate of lime, with or without the former salt: where both are associated, at first, the phosphate may disappear in a day or two, the oxalate remaining.* Albumen may be absent from first to last, appear temporarily, or exist in the urine of every twenty-four hours from first observation to convalescence,†—always in very small quantity. My observations prove that no connection exists between the appearance of albumen and convalescence.

The organic constituents generally range high, the inorganic salts fall below the average. Redtenbacher ascertained that, in particular, the chloride of sodium gradually diminished until hepatisation was established, when no traces of it could be found,—the salt reappearing with resolution of the disease.‡ Dr. L. Beale, further, finding an excess of the chloride in the sputa, refers the latter fact to a determination of the salt to the inflamed lung, depending on the activity of morbid cell-development occurring within it.§ It is, however, to be remembered that deficiency of common salt in the urine is by no means peculiar to pneumonia,—occurring in pleurisy, capillary bronchitis, rheumatism, and continued fever, endo-pericarditis, tuberculisation of the lung, &c. The nature of the food consumed at the time of examination must not be forgotten.

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* P. Kennedy, U. C. H., Males, vol. viii., p. 79, May, 1849.

† A. Bishop, U. C. H., Males, vol. ii., p. 194, July, 1847.

‡ Zeitschrift der Gesellschaft der Aerzte zu Wien, 1850.

§ Med. Chir. Trans. vol. xxxv., p. 325, 1852.

782. Among *cerebral symptoms*, the only one of frequent occurrence is cephalalgia; it comes on with the outset, as part of the febrile state. Delirium, coma, and convulsions are rare; complete insomnia is seldom observed. The *organs of sense* are not specially affected; epistasis, however, is sometimes met with.

783. *Prostration of strength*, as a rule, occurs from the first, and is so positive and so marked, that the fact may be made available in diagnosis; the exceptions are very rare. The *face*, more or less anxious in its expression, is of heightened colour generally, or particularly about the malar bones,—the tint being actually red or tending to lividity,—or pale, sallow, yellowish, earthy-looking.* When one malar surface only is red, this seems to depend rather on the decumbency of the patient, than on the lung affected. The *decumbency* is most commonly dorsal, with inclination to one or the other side. Andral affirms that not one out of fifteen patients lies on the affected side.

Terminations, Prognosis and Diagnosis.

784. The anatomical terminations of acute pneumonia are,—by resolution, diffuse suppuration, abscess, gangrene, and chronic induration; the clinical terminations, by recovery, death, and lapse into the chronic state.

785. *Resolution*, of which the signs have already been systematically set down, occurs at very various periods, and with different combinations of those signs. Thus, of 103 convalescents, observed by M. Grisolle, and discharged from hospital between the twentieth and fifty-fifth days of the disease, 37 had no morbid signs, 36 weak respiration, 14

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* Dr. Beale suggests that this sallow tint may be due to deficiency of chloride of sodium in the blood. Loc. cit. p. 369.

slightly blowing respiration, 11 redux crepitant or sub-crepitant rhonchus, and 5 deficient expansion with bronchitic rhonchi.

It is most important to observe that the physical signs of resolution, when the lung has been affected throughout by idiopathic inflammation, make their appearance first at the apex. If they pursue the contrary course, travelling from below upwards, the existence of tubercle superiorly is to be strongly apprehended.

Absolute fall in the frequency of the pulse and respiration, affecting, however, the latter in excess, whereby the pulse-respiration ratio returns somewhat nearer the natural standard, —diminished heat of skin,—with occasionally more or less marked changes in the urine as already described, are among the most striking general symptoms attending resolution.

786. What support does the doctrine of crises and critical days receive from the phenomena of pneumonia? Copious perspiration, cutaneous eruptions, hæmorrhages from the nose and kidneys, fœtid dejections, and deposits of urates, have been set down as critical occurrences. In respect of critical days, M. Andral refers to 112 cases, of which, it is affirmed, one half terminated on the seventh, fourteenth, or twenty-first days. In 34 cases, collected by M. Grisolle, where resolution occurred between the fourth and twelfth days, the fifth and ninth days are, on the other hand, the only ones exhibiting undue shares (9 and 11) of recoveries; the seventh day contributing only 3, or not quite 9 per 100 of the whole number.*

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* The whole subject of crises and critical days requires re-examination. Traube, who in his recent work (Ueber Krisen und kritische Tage,—or B. and F. M. C. Rev., 1853) refuses to admit crises of *diseases*, but acknowledges in a novel sense that of the *fever* accompanying them, appears to have struck out an important path of inquiry. This writer settles the question that has puzzled alike schoolmen and clinical observers from time immemorial, as to the relationship between the disappearance of fever and the occurrence of critical discharge, by cutting the Gordian knot, and affirming that sometimes the crisis is the effect, sometimes the cause, of the apyrexia!

787. The symptoms of *diffuse suppuration* are vague and unsatisfactory. Shivering may be completely absent, and dark fluid liquorice-juice expectoration, to which some writers attach much significance, may exist in the stage of red hepatisation. The general symptoms become more severe, and of adynamic character,—dry tongue, sordes on the teeth, pinched features, anxious expression, clammy skin, failure of strength of pulse, wandering delirium, or somnolence, and semi-coma occur. But all this *may* happen in cases where no suppuration has taken place; and as already admitted, there is no positively distinctive physical sign. Hence it follows, that the difficulty of proving the fact of recovery after diffuse suppuration, is extreme; in truth, there is no conclusive evidence of recovery having occurred in such cases.

788. There are no positive symptomatic evidences of the formation of *abscess*. The contents may be fœtid, from a sloughing condition of the walls. Such a case is readily to be confounded with primary gangrene of the lung. Abscesses may terminate favourably by passing into the state of quiescent cavity; and it is alleged (although they early—sometimes in a week—become lined with a pyogenic, and, eventually, pseudo-mucous membrane) by perfect cicatrisation.

789. The symptoms of *gangrene* of the lung will be separately considered presently, as likewise the subject of *chronic pneumonia*.

790. Dating *convalescence* from the fall of temperature of the skin, and restoration of the pulse-respiration ratio more or less closely to that of health, the process is generally rapid; the recovery of strength, and of such amount of flesh as has been lost, quickly follows. The ex-patient is for a time easily put out of breath, and often suffers from pain in the side, sometimes for many weeks. Percussion-dulness and various forms of morbid respiration gradually, but slowly, wear off. The signs of pulmonary œdema sometimes supervene.

M. Macario records two cases of pneumonia, in which, during the period of convalescence, formication and muscular weakness, commencing in the palms of the hands and soles of the feet, were followed by motor paralysis, perfect in the lower, imperfect in the upper limbs. The intellect was unaffected, and no excitement of the spinal cord observed. Of the real nature of the affection nothing is known: one case ended in recovery; the other in death, without *post-mortem* examination.*

791. The frequency of *relapse* has been estimated by M. Briquet at about one-fifth, by M. Grisolle at about one twenty-eighth, of the cases. The experience of this country supports the observation of M. Grisolle as to the rarity of true relapse, announced by fresh rigors, rusty expectoration and crepitation.

792. The *mortality* caused by pneumonia in Great Britain is very serious. Taking the estimates of the year 1839† as furnishing a fair average, we may rate the annual mortality in England and Wales at about 18,000,—an amount actually giving this inflammation the third place among fatal diseases. Further, out of 1,000,000 living population, 1201 annually fall victims to pneumonia.

Pneumonia destroys life, in London, much more extensively in the cold than the temperate months. It may be calculated from the invaluable returns supplied in the Registrar General's Twelfth Report, that in the metropolis the deaths in the quarter ending June, averaged 774; in that ending September, 477; in that ending December, 1185; in that ending March, 1250;—for the five years 1845 to 1849. The deaths average 2435 for the six coldest, 1251 for the six warmest months.

793. In fatal cases, death rarely occurs before the sixth, or after the twentieth, day. In the majority of instances, the patient perishes from gradual asphyxia; in rare instances from sudden obstruction of breathing through œdema of the glottis.

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* Bulletin Gén. de Thérapeutique, 1850.

† Second Report of the Registrar General. pp. 100, 154.

The rapidity of the fatal issue is by no means always proportionate to the amount of pulmonary tissue implicated.

794. The major influence regulating the *prognosis* of pneumonia in individual cases is *age*. In new-born infants, and after the age of seventy, the disease is almost always fatal; between the ages of six and twelve, death is the rare exception; between the sixteenth and twentieth years, scarcely one in fourteen of those attacked perishes; while in each succeeding decade up to the seventieth year, the deaths range between one-fifth and one-seventh of those seized. The disease, though destroying males more extensively than females, is, relatively to the numbers attacked, more fatal to females: the first of these propositions is based on the Registrar General's second Report, which gives 1339 male deaths, 1064 female deaths, to 1,000,000 of each sex living; the second on the results carefully obtained in France by MM. Briquet, Grisolle, and others. *Season*, if we are to trust M. Grisolle's experience, has but slight influence on the fate of those actually seized; of 100 pneumonic patients, very nearly the same number will perish in summer, and in winter: this result is rather startling, when compared with that we have just obtained, showing the influence of season in increasing the absolute mortality of pneumonia [792]; but the two are by no means irreconcilable. *Habitual drunkenness* and *weakness of constitution*, are of unfavourable augury. *Traumatic* pneumonia is, *per se*, the least dangerous. Inflammation of *both* lungs is more serious, as matter of experience, than of one; the *side affected* in single pneumonia does not appear to exercise any positive influence; but inflammation commencing with the *upper*, is more dangerous than that first implicating the *lower*, lobe. Above all, the so-called *epidemic constitution* is of signal importance in estimating the prognosis: at one period recovery is the rule, at another almost the exception.

According to Remak, the earlier the expectoration of fibrinous casts commences, and the more abundant and continuous it is, so much the more certain and speedy will be the cure. In fifty cases observed by him in Schönlein's wards, there were not more than four or five, in which even a partial diminution of the symptoms occurred previously to the appearance of the casts, while in the great majority an amendment was first observed after their occurrence.*

795. Pneumonia is very rarely immediately followed by tuberculisation of the lung: so rarely that intense predisposition to tubercle must be admitted, when such a consequence occurs. If lungs, already tuberculised, become acutely inflamed, convalescence from the pneumonia often takes place as rapidly, as if the lungs had previously been sound; and no increase in activity of the tuberculous disease necessarily follows. This fortunate course is, however, only observed, at least only habitually observed, where a small extent of lung has been inflamed: if the pneumonia be extensive, rapid breaking-up of tuberculised parts may follow.

796. The *diagnosis* of acute pneumonia as a rule is simple. The combination of crepitant rhonchus, tubular breathing, rusty expectoration, burning heat of skin, and perverted pulse-respiration ratio, is peculiar to itself. No one of these conditions can, however, be held to be pathognomonic of this disease; the greater the number of them associated in any instance, the more certain the diagnosis.

797. True crepitant rhonchus is distinguished from the fine bubbling, or sub-crepitant, of capillary bronchitis by its greater fineness, dryness, suddenness of evolution, and abundance. It exists at one base only,—the sub-crepitant at both; slight dulness under percussion soon attends true crepitation, and the signs of hepatisation quickly ensue; the pulse-respiration ratio in pneumonia is seriously perverted, while in capillary bronchitis it suffers, comparatively speaking, slightly; in the former the skin is acridly hot, in the latter moderately warm.

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* Diagnostische und pathogenetische Untersuchungen: Berlin, 1845; or B. and For. Med. Rev., April, 1847.

798. *Œdema* of the lungs is, except in very rare instances [206], distinguished by the comparatively large size and bubbling character of its rhonchus, by the absence of tubular breathing, by the deficiency of febrile action, heated skin, and perverted ratio of the pulse and respiration, and by the circumstances under which the disease originates.

799. Attention to the character of the different *pseudo-rhonchi* (pleural, mediastinal, and parenchymatous), as elsewhere described, will prevent their being mistaken for true crepitant rhonchus.

800. The distinctive marks of pneumonic solidification from a collection of fluid in the pleura, have already been given [688].

801. *Treatment.*—Acute idiopathic pneumonia holds a high position among those diseases of which the fitting treatment has been established by scientific experience.

In the first place, venesection has been numerically proved to diminish the mortality and lessen the mean duration of the disease, and also curtail the duration of its prominent symptoms, both subjective and objective,—the pain in the side, the febrile action, the peculiar expectoration, and the physical signs. Whether venesection possess the power of actually arresting the disease at the very outset, and preventing the occurrence of hepatisation, I hold to be yet scientifically undetermined; but that, in the immense majority of cases, it is vain to push bleeding to extremes in the hope of producing any such effect, clinical observation has amply proved.

In acute sthenic pneumonia, there are few barriers to venesection. Advanced age is none: Morgagni bled nonagenarians with success. Neither does pregnancy nor menstruation, provided the indications be otherwise positive, stand in the way of the use of the lancet. I have repeatedly bled women labouring under pulmonary inflammation, during the flow of the catamenia, without arresting the discharge; and if such stoppage should occur, cupping over the sacrum, or leeching the perinæum, will prevent any ill consequences. During certain epidemics, bleeding is very badly borne; and persons of a constitution shattered by excess, social anxiety, physical privation, or chronic disease, should, of course, be cautiously deprived of any of their, already spanæmic or hypinotic, blood. The earlier the bleeding, the better. M. Louis has shown that pneumonic patients, bled within the first four days, recover, *cæteris paribus*, four or five days sooner than those bled at a more advanced period; and Dr. Jackson, the enlightened practitioner of Boston, has proved that by bleeding on the first day, the mean duration, in a mass of cases at the Massachusetts Hospital, was lowered from 14·60 to 11 days.* No *period* of the disease is too late for blood-letting, provided the indication be thoroughly and strongly established on general principles. Even the *stage* of suppuration is by some held not to be a contra-indication, in itself alone, to the use of the lancet; but, although the name of M. Andral appears among those of the supporters of this doctrine, I have the strongest doubts of its correctness. M. Grisolle refers to four patients, bled to ten or twelve ounces, and in whom *post-mortem* examination, the sole positive test, proved the existence of the purulent stage. In all four cases, the fatal issue was obviously hastened, in one almost immediately caused, by the loss of blood.† No fixed rule can be laid down for the quantity of blood to be drawn; the mean amount of four pounds five ounces, taken from his patients by M. Bouillaud, has been most satisfactorily proved by M. Grisolle to have produced no more favourable immediate results (and of the ultimate ones what may not be feared?) than the abstraction of a mean quantity of two pounds seven ounces from a mass of patients treated by himself and others.* For my own part, I strongly question the utility of even such amount of depletion as this. Certainly, few cases have presented themselves to me in London practice, where it seemed necessary or advisable to draw blood oftener than twice; sixteen ounces sufficing in the first instance, and some ten or twelve in the second. Further, in cases of moderate severity, even in the male adult, abstraction of eight to twelve ounces from a vein, as the total quantity, effects all the good to be obtained by general bleeding. But I desire it to be understood that I only make this affirmation in regard of the sphere of practice with which I am familiar,—that of the metropolis. Slow convalescence is not the worst evil in cases where blood has been too lavishly sacrificed: a form of spanæmia is sometimes induced, which it may take months, nay years, to remove.

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* Putnam's Louis on Blood-letting. Boston, U. S., 1836.

† Sometimes, as we have seen [760], a lung apparently in a state of suppuration is in reality infiltrated with softened fibrinous exudation,—exudation cells alone, and no pus-cells, being found with the microscope. This anatomical fact, not generally known, may have some important bearing on the question of bleeding after the stage of red hepatisation has passed. But who shall distinguish, during life, the case of softened exudation from that of suppuration?

802. Leeching, or rather cupping, over the affected part, should always be employed in addition to general bleeding; in very mild cases it will suffice alone; local abstraction of blood affects pain much more directly and quickly than venesection. Six or eight ounces may, with propriety, be taken by cupping in a case of medium intensity, in addition to the quantity drawn from a vein; all local pain sometimes instantly disappears after the operation.

803. Tartarised antimony stands next in importance to blood-letting in the treatment of pneumonia,—were I, indeed,

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* Even in our own country, it was at one time thought by many that bleeding could scarcely be pushed far enough: men were systematically bled to convulsions. It was held theoretically sound to take away blood, the presumed source of the existing evil, to the uttermost point; but it was forgotten, or it was not known, that the increased impetus of the circulation during hæmorrhagic reaction might make up for the diminished quantity propelled. In those days, too, provided *theory* were satisfied, *facts* were held as matters of no importance. "Dr. Gregory, of Edinburgh," reports Dr. Watson, "used to bleed to the verge of convulsion. His colleague, Dr. Rutherford, seldom went beyond three bleedings, and generally accomplished his object by two. *His* patients recovered quickly; *Dr. Gregory's* very slowly." Yet Dr. Gregory continued to cling to his practice; for he had theory on his side.

henceforth, in the management of this disease, forced to surrender either, on the one hand, venesection, or, on the other, cupping and tartarised antimony, I should not hesitate to relinquish the former. In what manner this important agent produces its beneficial effects on the lung, is matter of the loosest speculation;—that it does produce such effects, is the really important point, and one of which scientific proofs abound. There is not any available evidence to show positively whether the effects of antimony on pneumonia are more marked when the mineral is, as is technically said, *tolerated* perfectly or imperfectly, or when it is not *tolerated* at all. The question could obviously only be decided by numerical comparison; and the number of cases in which complete tolerance is observed (that is, total absence of effects on the stomach and bowels) is relatively very small. Improvement often takes place within eight or ten hours after the medicine has been commenced with, and without any notable effect on the alimentary canal being noticed; whereas recovery also ensues when it acts freely both as an emetic and purgative. Hence it is more as a result of prejudice (for what but prejudices are even plausible *à priori* theories?) than of logical deduction from experience, that, in imitation of Rasori and Laennec, I prescribe antimony in such manner and combinations as are most likely to prevent its disturbing the stomach. The salt should at first be given in doses of half a grain, combined with dilute hydrocyanic acid, paregoric, and tincture of orange-peel, every hour for the first three or four hours,—and the dose then increased, at intervals of two hours, to one grain; in the course of twelve hours the quantity may be raised to two grains,—its repetition made less frequent, say every fourth hour.

804. The constitutional effect of mercury is by some held to be peculiarly efficacious in the stage of red hepatisation. It is even maintained that when that stage has been reached, calomel is a more valuable medicine than antimony. No scientific demonstration of this view exists. If it were correct, the

value of antimony in hospital practice, at least, would be singularly small; for the great majority of persons admitted into hospitals, have some amount of hepatisation, when first seen. Mercurials appear to me to be desirable in those cases of pneumonia only, where, for some cause or other, antimony is inadmissible.

805. Attended as it is, with the maximum amount of hyperinosis observed in any affection, pneumonia seems *à priori*, likely to be controlled by free and rapid administration of alkalies. Half a century ago, Mascagni treated the disease, during a severe epidemic, on this plan, with remarkable success; his follower, Farnese, gave the bicarbonate of potass in quantities varying from a drachm to an ounce daily.

806. Blisters are not advisable in the earliest periods of pneumonia: it would appear that they have no effect in shortening the mean duration of the disease, and they certainly increase fever and general irritation at the outset of the attack. At its more advanced periods, when fever has been materially controlled, they certainly relieve pain and dyspnœa, and seem to promote absorption of the infiltrated exudation.

807. The ordinary *juvantia* of the antiphlogistic regimen must, of course, be carefully put in requisition; the bowels, if necessary, should be opened by medicine; but profuse purgation, is, to say the least, absolutely useless.

808. Complete demonstration of the utility of treatment in pneumonia is found in the fact, that the mortality of the disease steadily increases with each succeeding day it has been allowed to run its course uncontrolled. The statistics of M. Grisolle, referring to the treatment by moderate bleeding and tartar emetic, show, that while the mortality among those seen and treated within the two first days, is only one-thirteenth, it rises among those whose treatment does not commence till the eighth day, from one-third to one-half of the whole number. Here, then, is a condition of success or the reverse, which should never be lost sight of in estimating the value of any

given mode of treatment. There are certain other circumstances, beyond the control of the physician, which exercise a most indubitable influence on the issue. Among these, the preexistence of organic disease, and the state of health generally of the individual, hold an important place. But of all collateral conditions, age is the most important: while at the two extremes of life, in the new-born infant and the octogenarian, pneumonia is, as we have seen, almost inevitably fatal; the mortality between the ages of six and twelve scarcely exceeds two and a half per cent. And if it be true, that between the ages of fifteen and thirty, the deaths equal about six per cent. of those attacked; suddenly rise to about fourteen per cent. in persons aged between thirty and forty; and thenceforth steadily increase with each succeeding decade; it is manifest, when we plume ourselves on the special excellence of a favourite system of medication, we should take count of the ages of our patients. There are periods of life at which it is next to impossible to save—there are periods of life at which it is not easy, with common prudence, to lose—a sufferer from idiopathic and sthenic pneumonia.

And hence it is, that, in spite of its apparent gravity, pneumonia is a disease which may, and actually has been, therapeutically played with, and this with impunity to the sufferers. Thus Skoda, drawing not a drop of blood, employing solely extractum graminis, or a few grains of nitre, and, in a few instances, corrosive sublimate, lost three only of forty-five patients; but the mean age of the series was only twenty-five and three quarters years.*

809. The value of the treatment of pneumonia by chloroform-inhalation, as practised by Wacherer, Baumgärtner, Helbing, Schmidt and Varrentrapp, must be as yet considered *sub judice.* Varrentrapp, one of its most earnest advocates, admits his want of perfect faith, for cases of severity were managed on other

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* Balfour's Report, Br. and For. Med. Rev., p. 591, Oct. 1846.

plans. Had severe cases been included, his mortality would have averaged about 11·5 per cent in a mass of patients aged thirty,* whose treatment commenced on the fifth day.

The number of inhalations averaged seventy-four in ten and a half days; the inhalation was not pushed to stupefaction,—the vapour of a drachm only on cotton being allowed to enter the lungs for ten or fifteen minutes; it was repeated every two, three, or four hours.

810. *Varieties.*—The varieties of pneumonia may be arranged in the following manner:

Varieties depending on—

(*a.*) Topographical seat . .			Single or double. Upper, lower, central, marginal, hypostatic.
(*b.*) Textural seat . . .			Lobar, lobular, interlobular.
(*c.*) Amount of subjective symptoms . . .			Obvious, latent, terminal.
(*d*). Secondary or intercurrent origin . . .	In new-born infants		Hardening of the cellular tissue, diphtheritic disease.
	From æt. 2 to æt. 16		Croup, cancrum oris, enteritis, measles, hooping-cough, variola.
	In adults .	Acute.	Rheumatic fever, puerperal fever, phlebitis, glanders, pyohæmia, peri- and endo-carditis, typhus, typhoid, febricula, acute diseases of the brain.
		Chronic.	Pulmonary tuberculisation and cancer, diseases of the heart, cancer of abdominal viscera, diseases of the liver, scurvy, purpura, Bright's disease, &c.

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* The age of three patients not treated with chloroform, but included here, is not given. Vide "Medical Times," Oct. 1851.

811. (*a.*) The topographical seat of pneumonia, though mainly interesting anatomically, is not devoid of clinical import, as a guide to the observer in quest of the physical signs of the disease. Of one thousand four hundred and thirty cases, seven hundred and forty-two were of the right lung, four hundred and twenty-six of the left, and two hundred and sixty-two of both organs. With respect to the cases of double pneumonia, which hold a rather high numerical rank (they furnish 18·3 per 100 of the whole series), it is to be observed that the great majority of them were not so from the outset; in other words, that the implication of the second lung was secondary in point of time. This, indeed, is a matter of no mean importance; for in doubtful cases the existence of the phenomenon at one only or at both sides of the chest, will aid materially in distinguishing the true crepitant rhonchus of pneumonia from the subcrepitant of capillary bronchitis. And even with the qualification now mentioned, alone, the frequency of double pneumonia is probably considerably exaggerated in the estimate just given: subcrepitation has often been mistaken for true crepitation, and a double capillary bronchitis put down as a double pneumonia; it is traditionally well known in Paris that even Laennec committed this error. The age of patients, too, must be borne in mind: in the adult, the proportion of double pneumonias does not probably much exceed one in twelve;* it has even been estimated so low as one in seventeen. On the other hand, the disease is almost always double in new-born infants; in one hundred and twenty-eight .such cases, observed by MM. Valleix and Vernois, the right lung alone suffered in seventeen cases, the left alone in no single instance; while both lungs were affected one hundred and seventeen times.

In two hundred and sixty-four cases, the upper lobe was affected one hundred and one, the lower one hundred and thirty-three, the middle part thirty, times (Grisolle). Pneumonia commencing about the middle of the lung is rarely primary: it is commonly either a sequence of endo-pericarditis or of blood origin,—a fact of obvious practical signification. The anterior *margin* of one or both lungs is sometimes separately inflamed: I believe that the frequency of this peculiar seat has been exaggerated from confounding mediastinal pseudo-crepitation with true pneumonic rhonchus.

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* In the scholar year 1834-5, when I was attending at the Hôtel-Dieu, 48 cases of pneumonia occurred in the wards of M. Choumel: 33 of these were of the right lung; 11 of the left; 4 were double.

812. Under the phrase "*hypostatic* pneumonia" have been described mere passive congestion of the bases of the lungs, occurring shortly before death, and also senile and adynamic inflammations.

813. Where the tendency to such congestion exhibits itself, occasional change of posture from the back to the sides, or even to the prone position, is one of the most important remedies; and, indeed, this is true, more or less, of every variety of pneumonia.

814. (*b*) The pneumonia of infancy very frequently, though by no means so constantly as is usually taught, instead of spreading through a lobe of the lung generally, limits itself to scattered groups of lobules, the intervening tissue remaining sound: such pneumonia is called *lobular*. So, too, pneumonia preceding the formation of secondary abscesses or collections of exudation-matter in the lungs, sequential to phlebitis, and pyohæmia, commonly assumes this form, no matter what be the age of the individual.

815. True lobular pneumonia is distinguished in the dead subject from collapse, and consequent solidity, of lobules, by the hepatised tissue being insusceptible of inflation; while merely collapsed nodules of tissue, as originally shown by Bailly and Legendre, may be blown up to their natural, or very nearly their natural, state. It is impossible to say in how many of the series of infantile pneumonias, above referred to, the real condition was nothing more than the collapse in question.

816. The physical signs of lobular pneumonia are obscure.

Inspection, application of the hand, and mensuration give merely negative results. Percussion, too, does not disclose such an amount of dulness as can be clinically trusted to; which is no more than might be anticipated, when we consider that the nodules of consolidated lung are separated by tissue perfectly permeable. In many cases originally to all appearance, at least, lobular, I have found the sound duller than natural, it is true; but when this was the case, and the opportunity of examining the parts occurred, I invariably discovered such extension of the inflammation between the nodules as to reduce the organ, physically speaking, almost to the state of ordinary consolidation. The respiration is exaggerated in some points; harsh, bronchial, or even slightly blowing (never tubular, so long as the pneumonia is simply lobular), in the spots probably corresponding to the consolidated nodules. Occasionally a few cracklings of an imperfect crepitant rhonchus may be heard; but it is difficult to distinguish these from the humid rhonchus of fine bronchitis,—a disease almost constantly associated in children with inflammation of the parenchyma.

817. The signs of *diffuse* pneumonia in the infant scarcely differ from those noticed in the adult. Crepitation, metallic tubular breathing, and dulness under percussion are the essential signs: the child's cry resounds with sniffling and bronchophonic character. The crepitation is of larger size than in the adult.

818. The *interlobular cellular tissue* may be the seat of acute suppurative inflammation,* pus occupying the situation that is filled by air in interlobular emphysema. Or this tissue may be infiltrated with fibrinous exudation, which solidifies into induration-matter, and causes considerable contraction of the lung, and sinking in of the side (Corrigan's " cirrhosis "). The bronchi undergo marked dilatation; and the pulmonary tissue, compressed by these tubes and by the surrounding induration-

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* Carswell's framed drawings, U. C. Museum, No. 57, C. b. 573.

matter, acting with its peculiar steady and constant force, becomes almost completely impermeable.*

819. The physical signs are flattening and diminished width of the side, impaired costal motion, increased vocal fremitus, percussion hard and dull or tubular, respiration irregular in distribution, weak, deep-seated, bronchial, or diffused blowing; while the rhonchi of bronchitis with hollow respiration indicate the existence of dilated tubes. The heart may, by this condition of things, be drawn to the affected side: as, however, there is very generally co-existent agglutination of the two laminæ of the pleura, it is difficult to say to what extent the state of the lung alone contributes to the displacement of the heart; such was the fact in the case of Osmond, referred to below.*

820. The distinction during life of this state of the lung from simple chronic pneumonia is always difficult, sometimes impossible. In "cirrhosis" of the organ, retraction of the side is, however, greatly more marked than in ordinary chronic inflammation; and if there be a considerable amount of flattening, we may be certain that it is not caused by the latter disease alone. The tubular percussion-sound, stronger respiration, signs of dilated bronchi, and traction of the heart towards the affected lung (only distinguishable, however, on the right side), met with in cirrhosis, are not observed in the simple inflammation.

821. (*c*) Instead of running the ordinary course with marked subjective symptoms, pneumonia may be completely *latent.* The perverted ratio of the pulse and respiration, and the physical signs, are then the sole guides to the detection of the disease. Pneumonia occurs in this form solely under circumstances of general physical debility: it is either *senile* or connected with *adynamic* diseases, of which it is an intercurrent phenomenon.

822. Physically, latent pneumonia is characterised by the

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* In a remarkable case of the kind (S. Osmond, U. C. H., Males, vol. iv., p. 336,) I found an infiltrated cellulo-fibrous induration-matter actually replacing certain lobules of lung: the pulmonary texture had been absorbed.

rapidity with which it runs into solidification, and with which it involves a great extent of substance.

823. In managing this form of the inflammation, the main attention must be given to the state of the system generally. Venesection I cannot believe to be ever requisite; and abstraction of blood, even locally by cupping, should be very cautiously ventured on. Still, if the respiration be much accelerated, and consolidation very rapidly extending, a few ounces of blood may be taken by cupping. Dry-cupping is always a measure of utility, and unattended with danger. The early application of blisters is by some observers strongly recommended in this variety of the disease: I have not happened to observe results justifying their confidence.

824. (*d.*) It is impossible to exaggerate the importance of pneumonia, when occurring as a *secondary* or *intercurrent* disease: in truth, the majority of cases of fatal pneumonia belong to this class. It is intercurrent pneumonia that commonly kills new-born infants, affected with hardening of the cellular tissue and diphtheritic disease. From childhood to puberty, croup, cancrum oris, measles,. hooping-cough, variola, frequently prove fatal through inflammation of the lungs. Again, we meet it complicating the diseases of the adult,—and if not at this period so frequently fatal, not the less important for the practitioner to watch. Thus it appears in continued fever, phlebitis, glanders, puerperal fever, inflammation of the bowels, and of the brain or membranes, and in acute rheumatism; among chronic diseases, in pulmonary tubercle, Bright's disease, chronic affections of the liver, not so commonly as might be expected in organic diseases of the heart, and in cancerous affections, not only of the thoracic, but of distant organs.

825. In treating intercurrent pneumonia, we must remember that the inflammatory character of the local malady is modified more or less seriously by the general state of the system. It is exceedingly probable indeed, that various differences exist

in the intimate constitution of many of the intercurrent pneumonias,—though at present no absolute proof of the fact can be given. Hence, if antiphlogistic management be proper, as it positively is in these cases, the state of the system at large should always be allowed full control. This is more especially true in the instance of diathetic diseases, such as rheumatism: it may be that colchicum is a more important remedy than antimony for rheumatic pneumonia. In pneumonia complicating purpura, the treatment, except in regard of blistering and dry-cupping, is wholly that of the blood-disease present.

CHRONIC PNEUMONIA.

826. Chronic pneumonia is rare as a sequence of the acute disease; it is rare as a primary disease; it is common as a local attendant on the progress of tubercle, cancer, and other adventitious products in the lung. I mean by chronic pneumonia, that form of disease in which impermeable pulmonary tissue is infiltrated with toughly-solid induration-matter, and in which there is no tendency to a softening process: these are its main characters. Circumscribed gangrene on a small scale occurs in very rare instances.

827. There is, however, also a form of disease in which the affected tissue is infiltrated with induration-matter, less tough in character, friable in short, and disposed to break up into a kind of granular detritus. This condition forms a kind of middle term between pure chronic inflammatory induration, and tuberculous infiltration.

828. When acute pneumonia lapses into the chronic disease, the strength and flesh, instead of returning with more or less rapidity, continue to fail; there is habitual, though moderate, dyspnœa; sensations of discomfort and oppression within the chest are almost constantly present; cough, with insignificant catarrhal expectoration, and unattended with hemoptysis, exists; there are thirst and anorexia, with irregular fever, which gradually grows constant, and has its evening-exacerbation,

rarely followed however, by any notable night-perspiration: with all this, the loss of flesh may, for a time, almost equal that occurring in the same period in phthisis.

829. Physical signs mark the changes in the lung: the surface is more or less extensively depressed, according to the area implicated; the chest movements are impaired, especially the costal ones; the antero-posterior diameter, and the superficial width of the side are diminished, and the percussion-sound is dull, sometimes wooden or tubular, with marked parietal resistance. The respiration is weak, uneven in quantity, harsh, bronchial, or diffused blowing in the affected parts,—occasionally exaggerated beyond these. The vocal resonance varies; it may be bronchophonic or null: the vocal fremitus is intensified. Chronic pneumonia has no rhonchus of its own; but there may be subcrepitation from bronchitis or œdema. In all probability under favourable circumstances, interstitial creaking-sound may be produced by forced inspiration in lung-substance in this condition.

M. Grisolle refers to a case observed by M. Requin, which shows that the auscultatory signs in chronic consolidation may be of a different kind,—in fact, all of them negative; total absence of all healthy or morbid respiration-sounds, of rhonchus, and vocal resonance, the percussion-sound at the time being completely dull. In the case referred to, the affection was mistaken for simple pleuritic effusion; but the patient dying *in a state of marasmus* two or three months after the outset of the affection, the sole morbid condition discovered in the chest was very firm induration, neither granular nor tuberculous, of the lower lobe of the right lung. Unfortunately we are not informed of the state of vocal fremitus. What the physical conditions were, capable of thus annulling the effects of conduction, unison-resonance, and echo, remains matter of pure conjecture; it is a parallel state of things to that sometimes observed in acute solidification.

830. In cases where the affection principally implicates the

upper lobe, and where obstruction of air-cells has been extensive, while the contraction of the exudation-matter thrown out into the substance of the lung has been active, flattening of the infra-clavicular region will take place. Under these circumstances, especially if, as often is the fact, among the general symptoms appear emaciation, and slow fever,—the distinction of the case from tuberculous consolidation is extremely difficult—impossible, indeed, unless by the aid of repeated examinations at certain intervals of time. The comparatively stationary condition of the part in simple consolidation, taken in conjunction with the progress of the general symptoms, if it do not perfectly explain the nature of the case, will, at least, point to the necessity of a cautious diagnosis. Fortunately difficulties of the class now especially referred to are of rare occurrence.

831. The treatment of chronic pneumonia is not essentially different from that of the early stages of tuberculisation. If the diagnosis were positively established, the occasional application of a few leeches, or the abstraction of three or four ounces of blood by cupping, might be more freely ventured upon, in the absence of all acute symptoms, than in phthisis.

The external application of strong ioduretted solutions, and the internal use of cod-liver oil, and bitter tonics, combined with alkalies, are medicinally the agents most to be trusted to.

ŒDEMA OF THE LUNG.

832. Œdema of the lung is anatomically characterised by infiltration of its texture with a colourless watery fluid, more or less completely airless; the organ is inelastic, pits under pressure and scarcely crepitates. The texture is unchanged, however; fragments, firm and resistent, sink instantly in water, after trifling, or without any, pressure.

A slight amount of pulmonary œdema is pretty frequently found in the body, no matter what have been the cause of death: clinically this is insignificant.

833. When of clinical importance pulmonary œdema is in the immense majority of cases a secondary state, either of passive or mechanical origin. Passive, when it forms a part of general dropsy, depending on morbid states of the blood, as in Bright's disease, purpura or scorbutus, or occurs in the course of acute general diseases, such as typhus or typhoid fevers. Passive, too, when it follows as a sequence of congestive conditions of the lung, as after the acute periods of bronchitis and pneumonia, or in the course of chronic bronchitis. Mechanical, when dependent on obstructive disease of the orifices of the left side of the heart, or on disease of, or extraneous pressure on, the pulmonary veins.

834. Hypertrophy of the right ventricle and pulmonary artery are conceivable causes of a sort of active œdema of the lung; but I have not met clinically with œdema of the sort.

835. Section of the par vagum induces, among other effects on the lungs, sero-sanguineous infiltration of their texture; but of the influence of morbid states, dynamic or statical, of the nerve in producing such œdema, nothing is known but by conjecture. Is the effect in vivisections of passive or active mechanism? Laennec taught that pulmonary œdema may occur as a primary and idiopathic condition, and that the suffocative orthopnœa, which sometimes cuts off children after measles, arises from such œdema.

836. In cases of general dropsy the external anasarca, as is well known, seems to be rapidly translated in some cases to internal cavities, especially the peritonæum, more rarely the pleura. I have once or twice known the transference take place especially to the parenchyma of the lung.

837. Considering the numerous ways in which pulmonary œdema is speculatively producible, it is of rare occurrence.

838. Œdema, under whatever conditions generated, is usually either diffused through the lungs or limited to those parts, where the laws of gravitation would naturally direct the fluid.

But serosity may accumulate especially at the apices. In a case of emphysema,* carried to so high a point that the anterior edges of the lungs overlapped superiorly, and almost completely covered the heart inferiorly, and in which there were dilated bronchi, bronchial abscesses, and some spots of pulmonary apoplexy, the apices, bearing the marks of the ribs upon them, were loaded with black-coloured fluid, airless and of watery consistence. There was no disease of the mitral orifice or of the right side of the heart: nor was there any obvious obstruction of the pulmonary veins.

839. Disturbance of respiration from a slight to an intense degree, slight cough, watery, or sometimes rather tenacious, expectoration, sensation of weight and heaviness within the chest, constitute the symptoms—a combination anything but distinctive: neither are the physical signs conclusive. Inspection discloses nothing sufficiently marked to be trusted to; the vocal fremitus may be slightly intensified; the percussion-sound is duller than natural; the parietal resistance increased; the vocal resonance varies in character; the respiration is weak, and harsh, or even blowing, and mingled with fine bubbling rhonchus, or in rare cases with a rhonchus not distinguishable from true crepitation: the fine bubbling rhonchus, when very liquid and well marked, is the most distinctive sign.

840. In congestion of the lung, if rhonchus exist, it is drier than in œdema, the expectoration more viscid, and there are no dropsical symptoms. Hydrothorax is unattended with rhonchus, and the dulness, caused by the pleural fluid, changes its seat with the posture of the patient. Pleural pseudo-rhonchus, unless care be taken, may be confounded with the bubbling rhonchus of œdema. The rhonchus of capillary bronchitis is rather to be distinguished, it must be confessed, by coexistent evidence of bronchial inflammation, than by its own characters.

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* Skinner, U. C. H., Males, vol. ix., p. 97; the right lung weighed 42½ oz.; the left, 37½ oz.

841. Œdema, occurring after pneumonia, furnishes an indication for the use of gentle tonics. If it form a part of general dropsy, it is mainly to be relieved by means calculated to lessen the latter. But dry-cupping and a succession of flying blisters to the chest, sometimes exercise a distinctly beneficial local effect.

GANGRENE OF THE LUNG.

842. Laennec described two forms of gangrene of the lung, the diffused and the circumscribed: the tissue may besides sphacelate in patches or in nodules, embracing a greater or less number of lobules. Of sixty-eight cases, sixty-two are represented to have been of the circumscribed, six of the diffused form.*

843. In the diffused form there is no evidence of limitation, either by exudation-matter or otherwise, of the gangrenous process. In the circumscribed form, the sphacelated parts are surrounded by tissue infiltrated by exudation-matter of low plastic type. In both forms excavation occurs; in both perforation of the pleura, of the œsophagus, and even of the diaphragm may ensue; fatal pneumo-hydrothorax may thus be caused; or, both pleural layers having been previously agglutinated, may be perforated, and the gangrenous ichor conveyed beneath the skin,—subcutaneous emphysema following. Hæmorrhage, either into the bronchi or pleura, profuse enough to cause instant death, has sometimes occurred from perforation of a vessel; but this is rare, inasmuch as both arteries and veins are commonly plugged with coagula not only in, but on the confines of, the mortified tissue. The bronchi are cut abruptly across by the sloughing process. An observation by Schroeder van der Kolk,† in which he found the pulmonary lymphatics and the bronchial glands more or less gorged with gangrenous fluid, requires confirmation: though there can be little doubt of

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* Dict. de Médecine, t. xxii.

† Obs. Anat. Path., t. i.

its correctness, both from the character of the observer, and the fact that purulent matter distinctly makes its way from abscesses in the lung to the lymphatic vessels.

844. Pulmonary gangrene is so commonly fatal, and fatal in so short a period, that opportunities rarely occur of tracing the changes undergone by the excavations, it effects. There is evidence, however, to show that a cavity of the sort may eventually become lined with a pyogenic, or pseudo-mucous, membrane, secreting a fluid wholly free from gangrenous odour. In a case that has been under my observation for a considerable period, and which I believe to be of this kind, the sputa temporarily acquire a putrid smell from time to time,—but whether from mere alteration in quality of the secretion, or from patchy sphacelation of the surface of the excavation, I am unable to say.

A tendency to cicatrize is in very rare instances exhibited by these cavities: but of actually complete closure I have met with no recorded example. A case described by Dr. Gerhard shows that nine years after the formation of a gangrenous excavation, closure may not be effected.

845. Cruveilhier describes an odourless variety of gangrene: the texture falls into shreds; no smell is observed either during life or after death.

846. The diffused and circumscribed forms are generally limited to a single spot; nodular sphacelus, like pyohæmic abscesses, frequently affects many together. The periphery of the lungs seems especially to suffer from the latter, the postero-inferior and central parts from the former. The right lung is more commonly affected with diffused gangrene; both organs are implicated, when the disease is nodular.

847. Pulmonary gangrene is either solitary, or associated with similar change in various parts. The former is more common in the adult; the latter in infancy. In certain hæmic diseases, too, several parts or tissues suffer, coetaneously or consecutively.

848. The conditions, under which gangrene of the lung occurs, may be thrown into tabular form, as follows :—

Gangrene of Lung produced by—

I. Local diseases .	Pneumonia, acute and chronic; tuberculisation; cancer: hæmorrhage; hydatids.	
II. Cardic disease .	Acute endocarditis of right heart ?	
III. Hæmic diseases .	Animal venoms; stings of certain insects.	
	Morbid viruses	Glanders; exanthemata; typhoid fever; purpura; scurvy; pyohæmia; gangræmia.
	Heteræmia; poisonous gases.	
IV. Perverted innervation of the lungs . .	Epilepsy; insanity; organic cerebral disease.	
V. Traumatic.		

849. I. The rarity, with which acute pneumonia eventuates in gangrene, has already been insisted upon [772]: where such termination does occur, it may be immediately determined by excessive violence of the inflammation, or by obstruction of nutrient vessels; under either circumstances a depraved state of the blood is, with general adynamia, probably necessary as a prior condition. In rare instances the vascular obstruction caused by the progress of tuberculization and of cancer, by copious infiltration of blood, and the pressure of a hydatid sac, have all been productive of local gangrene on the same principle.

850. By the obstruction of what vessels is the failure of nutrition brought about? Sir R. Carswell thinks by that of the pulmonary artery. Of the obstruction of the contiguous branches of that vessel, which he has figured, and which I have also seen on an extensive scale, there can be no doubt. But physiologists assign the nutritive quality especially to the bronchial arteries; while the blood of the pulmonary artery can

only nourish, it is held, after oxygenation in the capillaries and to a limited extent: now the state of the bronchial arteries in gangrene has never, that I know of, been examined.

851. II. May obstruction of minute branches of the pulmonary artery, from circulation of the products of endocarditis of the right heart, cause patches of gangrene in the lung? All I know on the matter is this. In a child, cut off by endo-pericarditis affecting both sides of the heart, and central pneumonia of the left lung, there were three or four pea-like spots in the inflamed tissue distinctly gangrenous: the condition generally of the child was not favourable to gangrene; and it seemed possible that plastic products or minute fibrinous concretions from the endocardium might have plugged the capillary vessels. Such facts, if not otherwise explicable, would tend to prove that the nutrition of the lung is really in part effected by the pulmonary artery: they would also corroborate Dr. J. Taylor's views on the mechanical production of pneumonia by endocarditic secretions.

852. III. Sir R. Carswell figures gangrene of the lung, associated with that of the lip, ensuing on the bite of an insect. In children gangrene of the lung has particularly been noticed after measles, scarlatina, and variola, commonly associated with cancrum oris; so, too, in the course of typhoid fever, purpura, and scurvy; in rare cases of glanders; in the purulent, and in the gangrenous* diathesis, the lung sphacelates in a certain proportion of cases. Poisoning of the blood by the gases evolved in animal putrefaction, seems to cause pulmonary gangrene in some persons. The slow deterioration of the vital fluid worked out by habits of debauchery and by privation seems to be the groundwork of gangrene in many instances, if not to directly produce it. In all cases of this class there is a tendency to hæmorrhagic effusion and passive congestions.

853. IV. The occurrence of gangrene of the lung has been

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* A diathesis which, though not generally recognised, is just as real as the purulent, and for which the name gangræmia might be adopted.

noticed by many persons as a dependance on cerebral disease. Of the tolerably frequent concurrence of the two states, there can be no doubt: and the former seems fairly explicable by deficient or perverted nervous influence, just as the local gangrene, which sometimes occurs in a hemiplegic side.*

854. V. In those rare instances where the lung has mortified after external injury to the chest, some favouring condition of the blood in all probability pre-existed.

855. Sphacelation of the lung occurs at all periods of life; the subjoined table, constructed from his own observations by M. Ernest Boudet,† furnishes by far the most trustworthy information on this point, as well as upon the frequency of the disease, and, indeed, of spontaneous gangrene in general.

AGE.	Numbers of post-mortem examinations.	Cases of gangrene of the lung.	Ratio of cases of gangrene to post-mortems.	Cases of various spontaneous gangrenes.	Total cases of gangrene.	Ratio of cases of gangrene to post-mortems.
Children . .	135	5	1 : 27	9	14	1 : 9
Adults . . .	156	2	1 : 78	4	6	1 : 27
Aged persons	220	2	11 : 19	7	9	1 : 24
	511	9		20	29	

856. The *symptoms* in the *diffused* form are great general prostration, oppressed breathing, profuse expectoration, frothy and purulent-looking, of gangrenous odour, with a small, feeble, and very frequent pulse, and all the general appearances of intense adynamia. The power to expectorate is soon lost, and death directly occurs from suffocation.

857. The course of *circumscribed* gangrene is somewhat different. At first the evidences of affection of the lung are commonly extremely obscure; the signs of pulmonary congestion

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* Clin. Lect., "Lancet," loc. cit., p. 281.

† Archives de Méd., Sept. 1843.

exist, coupled with an amount of prostration quite out of proportion with the extent of local disease; the expectoration muco-purulent, rarely bloody in adults, frequently so in infants and children (if they expectorate at all), acquires, the moment perforative communication is established between the bronchial tubes and the gangrenous tissue, properties more or less strongly characteristic of the disease. It is of dirty-greenish, yellowish-brown, or ash-gray colour; very liquid; and exhales an odour distinctly gangrenous, or resembling that of wet plaster, or *sui generis* but painfully fœtid. In the adult the breath generally has the same fœtor, but this may not be constant: the expired air may be completely free for some minutes at a time from disagreeable smell; when suddenly, without cough or any other apparent cause, it becomes intolerably fœtid.* Possibly temporary plugging of the bronchi communicating with the mortified tissue may account for the absence of odour. In children, fœtor of the breath is less common than in adults,—occurring in scarcely a third of those affected.

858. The physical signs are those of consolidation followed by those of breaking up of tissue and eventually of excavation. There is no special character in the signs of any one of these conditions indicating the nature of the disorganising process. The signs of pneumonia may precede, and those of bronchitis accompany, the evidences of giving way of tissue.

859. The terminations of circumscribed gangrene are by death or recovery. Death may occur rapidly by collapse, or sometimes by hæmoptysis or hæmothorax. Or the fatal event may take place slowly: abundant purulent fœtid expectoration, hectic fever, night sweats, emaciation, wear out the patient; and, after protracted suffering of weeks, or even months, death ensues. In cases of recovery (unfortunately a small minority), the discharge loses its fœtor, diminishes in quantity, and becomes simply mucous; the hectic fever ceases, the appetite returns, and a complete rally is gradually accomplished. The

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* D. Hayes, U. C. H., Males, vol. vi., p. 92.

physical signs of local consolidation, deficient breathing-action, and sometimes of excavation, remain.

860. The diagnosis of gangrene of the lung turns upon the peculiar fœtor of the breath and expectoration, coupled with the physical signs of softening and excavation of the pulmonary substance, ensuing upon those of sub-acute adynamic pneumonia, or upon those of congestion of the lung arising in the course of the various morbid states already enumerated. But there are sources of fallacy of two kinds: there may be the extremest fœtor and profuse greenish sero-purulent expectoration, and yet no true gangrenous cavity formed; and there may be a gangrenous cavity without the characteristic conditions of the breath and sputa.

861. In the first class of cases appear examples of acute added to chronic bronchitis, where the breath and sputa acquire gangrenous odour, without any evidence of excavation,—where recovery takes place, the fœtor being merely temporary, and where the only plausible explanation of the facts is, that sloughing of the mucous membrane has occurred within the tubes on a small scale. I have no proof of this view, however, to offer in the form of *post-mortem* examination. Again, the breath and sputa sometimes acquire gangrenous odour, in cases of broncho-pleural fistula with empyema, where changes in the pleural sac are the real causes of the fœtor. I have even known the expectoration acquire that odour, in cases of non-communicating empyema, apparently from chemico-vital changes in the pleural cavity,—a fact assimilable to that, familiar to surgeons, of the stercoraceous odour sometimes acquired by the contents of abdominal abscess without intestinal communication. In both cases transudation of fœtid gas through softened and rarified tissue of low vital resistance, probably occurs.

862. But even the positive signs of cavity in the lung *plus* the fœtor, do not prove that the cavity is of essentially and primarily gangrenous nature. It may be tuberculous, or it may be purulent. I have now seen some half-dozen cases

of consumption, in which the special fœtor occurred incidentally in connection with tuberculous cavities already formed. In one of these instances (Consumption Hospital, Chelsea), the expectoration of a fœtid pea-like mass, distinctly possessing microscopically, and even to the naked eye, the characters of pulmonary tissue, put a term to the gangrenous discharge,—a fact which I hold to be proof positive that a minute sphacelated spot may impress the characteristic fœtor upon the expectoration and breath, quite as effectually as gangrene of extensive area. A tuberculous cavity, thus locally gangrenous, is very difficult to distinguish from true gangrene of the lung, if the case be seen for the first time when that change has occurred, and if the history of the case be imperfect. The seat of the cavity at the apex, and the existing signs of induration at the other upper lobe, once guided me successfully to the diagnosis in a case of this kind; but I am far from thinking the observer would always be justified in an absolute affirmation on such data. A fœtid abscess is generally distinguishable from true gangrene, not by the amount of fœtor, which may be just as great from local sloughing of the walls of the abscess, but by the fact that the signs of excavation precede the occurrence of fœtor in the case of abscess, follow it in that of true gangrene.*

The second variety of case, where real gangrene occurs without perceptible fœtor, is rare, especially in the adult. In children a guide to the diagnosis of an excavation thus formed, is sometimes found in hæmoptysis; for, singularly enough, while tubercle scarcely ever causes hæmoptysis in childhood, gangrene is then frequently attended with that symptom.

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* In a case which fell recently under my observation (Dl. Hayes, U. C. H., Males, vol. vi., p. 92, 1851), there were occasionally such abundant discharges of almost pure pus, alternating with the more characteristic diffluent gangrenous expectoration, that, had not fœtor preceded by some days the signs of softening and excavation, I should have been disposed to regard the case as one of fœtid abscesses successively bursting, and not of gangrene: unfortunately permission to examine the body could not be obtained.

863. Gangrene of the lung-substance, or of induration-matter infiltrating it, may run a chronic intermitting course. At least, I have seen a case in which fœtid gangrenous expectoration continued with intermissions for months, without much attendant constitutional suffering, but with gradual development of the signs of excavation in a spot where, I was assured, the existence of a gangrenous cavity had been affirmed by a practised observer some time before, but of which no positive evidence remained, when I first became acquainted with the patient. Had accumulation of induration-matter taken place here, and subsequently slowly sloughed away?

864. The condition referred to a moment since as connected with endocarditis [851] may be considered an example of nodular gangrene; I have also seen it in one case of so-called secondary abscesses; only a certain number of the nodules, and only the central part of these, were actually sloughy.

865. The prognosis of gangrene of the lung cannot be held to be absolutely fatal. Recovery has been calculated to occur in one twelfth of cases: this, however, I regard as altogether too high an estimate of the favourable chances, unless it be understood to include the most trivial cases, as, for instance, partial sloughing of the walls of a cavity. It must be remembered that the characteristic fœtor may be impressed on the sputa by the merest fragment of mortified tissue.

866. In the *treatment* of gangrene of the lung, the chief reliance has hitherto been placed, in the acute state, in stimulants and tonics: the sesqui-carbonate of ammonia, opium, and camphor, in various combinations with bark, or quinine, given in full and repeated doses, are held to afford the patient the best chances of recovery. The effect on the powers of the system generally, often produced by the first few doses, is really extraordinary. Wine or brandy may be administered at the same time.

The remarkable effects of chlorate of potass in gangrene of the mouth entitle this salt to a trial, where the lungs suffer;

the effects of yeast in frequently repeated doses of an ounce, also deserve investigation. I should be disposed to confide more in either of these agents than in ammonia, the permanently beneficial effects of which I have never, I confess, seen demonstrated.

867. The only local measures advisable, under ordinary circumstances, are dry-cupping and counter-irritation by blistering or otherwise; and these only when the disease appears, in the main at least, of local origin. If, however, the evidences of acute secondary pneumonia, produced by the irritative action of the sloughed lung, are conclusive, and the system generally has rallied, a few ounces of blood might be cautiously abstracted by leeches or cupping, from the affected side: the positive indication for this practice will, however, very rarely arise; and has never done so within my own experience.

868. In cases lapsing into the chronic state, the mineral acids and quinine become the main remedies; of the former, the nitro-muriatic is probably the best.

869. Fœtor should be corrected by chlorinated mouth-washes, or fluids containing creasote in suspension. The chloride of zinc may be used for this purpose in a state of extreme dilution; three grains to eight ounces of water. Inhalation of tar vapour, of creasote, of chlorine, should be had recourse to, not only as corrective of fœtor, but as tending, in all probability, by their direct chemical action on the sphacelated tissue, to control septic changes within the lung, and so lessen the local irritation, and constitutional depression.

870. The diet should be nutritious and digestible: strong beef-tea, thickened with isinglass or prepared gelatine, finely-pounded meat, eggs beaten up with small quantities of brandy, milk, &c., should be given as frequently as the digestive powers of the patient appear to permit. Good porter in moderation is an advisable beverage.

PULMONARY HÆMORRHAGE AND HÆMOPTYSIS.

871. Hæmorrhage may occur from the mucous membrane, or other actual tissue, of the tubes; or, within the tissue of the lung itself; it is, accordingly, either bronchial or pulmonary.

872. *Bronchial.*—It is generally stated in systematic works that bronchial hæmorrhage is extremely frequent; but, if the term be understood strictly in the sense above given it, I cannot help believing that it is of extraordinary rarity. Setting aside those instances—mere curiosities from their singularity—in which ulcers in the bronchial tubes, or plastic bronchitis, furnish the blood of hæmoptysis, I have never yet seen a case where blood discharged during life even appeared, much less was proved, on inspection of the bronchial tubes, to have come from their substance by molecular ruptures, the exhalation of the older writers. True, there is no *à priori* reason why blood should not ooze from the bronchial mucous membrane, as we know it does, as we actually sometimes see it do, from the mucous membrane of the mouth: but, on the one hand, the evidences of the occurrence are wanting; and on the other, it is found in the great majority of cases (there are many in which it is impossible to form an opinion on the point), that, when blood has made its way into the bronchial tubes, the pulmonary tissue itself is the source of supply. In cases of diseased heart attended with hæmoptysis during life, there may always, as far as I have examined, be found more or less marked evidence that the parenchyma of the lung, and not the mucous membrane, has given way molecularly: the evidence I refer to, is the presence of dark blood-points here and there in the pulmonary tissue—a sort of embryo pulmonary apoplexy. While, on the other hand, I have often found the finer tubes, as far as they can be followed with a scissors, free from undue vascularity or marks of saturation with blood,—a fact the

more remarkable, because the larger trunks are occasionally, in such cases, very evidently imbibed with that fluid.

These remarks are, however, not to be understood to apply to escape of blood on a microscopical scale; and I fully admit that the whole question involved requires closer investigation than it has yet received. Ecchymoses and petechiæ of the bronchial walls occur in scurvy and purpura.

873. When blood *is* furnished by the bronchial tubes, hæmoptysis is its symptom, and thin bubbling abundant liquid rhonchus in the bronchi its physical sign. It does not appear to stagnate sufficiently within the tubes, when of this origin, to alter the percussion-sound.

874. *Pulmonary.*—Pulmonary hæmorrhage, that is hæmorrhage springing from the actual parenchyma, occurs with or without recognised anatomical characters.

875. In the first class of cases, we meet with the nodular and the uncircumscribed pulmonary apoplexy of Laennec, petechial, and, lastly, interlobular hæmorrhage,—an example of which hitherto undescribed form I once observed in a new-born infant. No one of these anatomical states is necessarily productive of, or connected with, hæmoptysis.*

876. On the other hand, there is no established morbid anatomy of the most frequent variety of hæmoptysis, that depending on the process of tuberculisation. Molecular ruptures of the capillary vessels of the parenchyma are doubtless the cause of the discharge of blood in tuberculous diseases, except in those very rare cases where a vessel of some size is perforated; but absolute demonstration of the fact is still a desideratum.

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* It has been shown, indeed, by Dr. Watson, that nodular apoplexy may sometimes be an *effect* of certain conditions, producing discharge of blood through the mouth, instead of being the cause of the latter. In a man dying from hæmorrhage from the lingual artery, several apoplectic nodules were found, evidently formed of blood, which had trickled downwards from the mouth through the windpipe, and accumulated in the air-cells.

877. In the circumscribed variety, the blood, contained in the air-cells, forms sharply-defined, rounded, nodules, from half an inch to four inches in diameter, excessively firm to the feel, and raising the pleural surface, if situated close to this. On section these nodules are of very dark red venous hue; slightly, or not at all, granular; homogeneous-looking, except that in rare instances clotted blood in small quantity may be found in the centre,—in these latter cases only is blood expressible in any quantity; commonly seated rather in the lower and posterior parts of the organ than elsewhere; varying in number from one or two, to some twelve or fifteen, five or six being about the average, and in the great majority of cases affecting both lungs, though in different degrees; and bounded by tissue, either quite natural, or slightly darkened by blood, apparently imbibed from the periphery of the nodules.

Gradually the outline grows less sharply defined; the tint lightens, by passing into brownish, and eventually yellowish red; the lung-structure reappears, as absorption advances; the blood-vessels and bronchi, at first impermeable, allow water and air to pass; the seat of old hæmorrhage is tough.

In very rare instances suppuration or gangrene occur. It is said the effused blood sometimes becomes surrounded with a cyst; and also that pseudo fibro-cellular formation may arise within it. I have never seen either condition.

Clots have been found both in the pulmonary artery and veins; it is probable the capillary vessels supply the blood.

The adjoining texture is sometimes inflamed,—the inflammation being coetaneous, or consecutive, not preparatory.

878. In the diffuse variety the pulmonary texture is torn: blood infiltrates the inter-vesicular tissue; the pleura may undergo rupture, and instantaneous death from abundant hæmorrhage ensue. Encephaloid cancer sometimes furnishes the blood in this variety,—which, as far as I have seen, is decidedly rare.

879. An infant, born to all appearance healthy, breathed

well for two hours, without exhibiting discoloration of the skin, or other morbid appearance. Sudden dyspnœa then came on; the accoucheur, being still in the house, ran up stairs; on his arrival the infant was dead. I found the lungs gorged with blood, except at the anterior border; between the lobes and lobules lay dark clotted blood in considerable quantity. There was much vesicular emphysema with subpleural vesicles, and air appeared in some points between the lobules.

880. Petechial hæmorrhage occurs in the lung-substance in purpura, scurvy and hæmorrhagic exanthemata.

881. Pulmonary apoplexy, in the nodular form, is almost invariably an effect of disease of the heart, especially of the mitral orifice: for my own part I have scarcely ever seen it, except in cases of mitral disease.* It sometimes occurs, we are assured, from a "diseased state of the pulmonary vessels and parenchyma," independently of heart-disease. Of diffuse hæmorrhage few examples have fallen under my notice: cancerous disease of the lung, or injury to the chest, were the immediate causes.

882. The symptoms of pulmonary apoplexy are exceedingly difficult to specify, because they are mixed up with those of pre-existing disease in the heart, and of disturbance, secondary to this, in the lungs. Dyspnœa, tightness, and dull pain in the chest, all exist independently of such apoplexy in mitral disease; they, however, increase in severity when blood escapes into the lung-substance, as does likewise any cough previously present. The only symptom really important is hæmoptysis, in the forms of tinged mucus, striæ of blood, pure blood, rarely florid,

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* In a case recently observed (Fosbury, U. C. H., Males, vol. x., p. 133) of great hypertrophy of the left ventricle, the mitral orifice, almost circular in shape and gaping, readily admitted the points of the thumb and four fingers of a medium-sized hand: the valve and cords were texturally sound. I supposed the valve might not have been large enough to close the dilated orifice; but found that on tying the aorta, and projecting a column of water into the ventricle, through the opening made by cutting away the apex of the heart, none of the water made its way into the auricle. Hence there had been no mitral regurgitation, yet there was slight pulmonary apoplexy.

rather darkish, of bistre tint, or even sooty-looking. The quantity of blood is habitually small; and I have never once, out of a very considerable number of cases, known anything like profuse hæmorrhage attendant on nodular pulmonary apoplexy. I should not venture yet to say that there is any special appearance in the blood positively distinctive of its apoplectic origin; still my memory does not supply me with a single instance of bistre or sooty-coloured blood coming from the lungs, except in cases of the sort. Where such sanguineous expectoration has ceased before death, the nodules have exhibited signs of absorption. It is to be remarked that red discs may often be found in the sputa of persons with mitral disease, where there is no blood visible to the naked eye.

883. The physical signs of nodular hæmorrhage are, also, very obscure. The effusion of blood must, to say the least, be rarely sufficiently great to act as an impediment to chest-motion; such effect has certainly never fallen under my notice.

If the nodules be few and small, percussion and auscultation give no positive information. But under the converse circumstances, I have known the vocal vibration somewhat intensified over the nodular masses, and the percussion rendered very notably dull. Over the accumulated blood the respiration is weak; beyond it, harsh, bronchial, or diffused blowing; the state of vocal resonance varies. If there be hæmoptysis, thin, liquid rhonchi will be present. Even within the apparent area of the apoplectic nodules, fine bubbling, almost crepitant rhonchus, may sometimes be caught on full inspiration; but it would be impossible to prove that the rhonchus ever really originates within the nodules. Instead of undergoing gradual absorption, pulmonary apoplexy may act as a source of irritation; the signs of local pneumonia, abscess, and, in rare cases, even of gangrene, may then be successively noted.

884. In regard of treatment, assuming that there are no signs of copious discharge of blood, I am disposed to insist most strongly on the value of extensive and repeated dry-

cupping of the chest. I have repeatedly known hæmoptysis stopped almost at once by this measure, in cases of mitral disease, when the general aspect of the patient forbade the abstraction of blood. A small quantity of blood may be removed with propriety, where there is no prominent asthenia. Counter-irritation, by blistering and otherwise, and free purgation, are the next most important remedies. The treatment must always be controlled by the state of the heart and of the secondary disorders dependent on that organ. Unless the hæmoptysis be considerable, it is not advisable to employ the ordinary astringents; digitalis will be useful or detrimental according to the condition of the heart's substance and orifices, a matter to be discussed hereafter.

HÆMOPTYSIS.

885. Under the title of hæmoptysis, or expectoration of blood, may be included all instances of discharge of that fluid, from, or through, any part of the air-passages below the epiglottis. The quantity of blood may be so small as to be barely visible to the naked eye, or it may amount to pints, or even quarts.

886. Hæmoptysis occurs sometimes in the ascent of lofty mountains; its occurrence is then commonly referred to diminished pressure of the atmosphere. It may follow wounds of the lung and injuries of various kinds to the chest; and it may take place *vicariously*, as a periodical discharge in females, instead of the catamenia. Except under these circumstances, whatever be its immediate and direct mechanism, hæmoptysis is a sign of disease of the air-passages, lungs and appendages, or of the heart or great vessels.* It may be that in certain instances

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* Carcinomatous disease of the œsophagus may lead to spitting of blood; I have not actually observed this, except where the air passages were perforated, but conceivably it might occur independently of this condition.

of scurvy, purpura, malignant typhus, and the hæmorrhagic exanthemata, slight escape of blood through the bronchial tubes occurs; but I know, that even in the worst cases of the kind hæmoptysis may be totally absent.*

887. The diseases which act as more or less frequent causes of hæmoptysis are, in the:—

Larynx: ulceration phthisical, cancerous, syphilitic;
Trachæa: ulceration;
Bronchi: bronchitis, simple and plastic, ulceration, cancer;
Bronchial glands: tubercle, cancer;
[illegible] Lung: congestion, active, passive, and mechanical; pneumonia, acute, and chronic; abscess; gangrene; tubercle; cancer; hydatids;
Mediastinal tumour;
Heart: mitral disease; hypertrophy of the right ventricle; dilatation with feebleness of the left ventricle;
Great vessels: aneurism.

888. But, although it is impossible to ignore in pathology the power of all these affections to produce hæmoptysis, in actual clinical practice the symptom is so frequently connected with tuberculisation of the lung, that it comes to be one of its most significant symptoms. The laws of tuberculous hæmoptysis will hereafter be considered. In the present place, I shall simply place before the reader an analysis of the clinical evidence I have been able to collect, illustrating the closeness of connection between hæmoptysis and pulmonary tubercle in the adult.

889. "The *quantity* of blood voided, is the first point for consideration. It is commonly said that the expectoration of streaked or tinged sputa is utterly insignificant, because such are seen in *bronchitis;* but no attempt has ever been made to decide numerically to what extent this is true. I find that in twenty-

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* C. Lennell, U. C. H., Females, vol. ii., p. 217. Hæmorrhagic variola and scarlatina combined; hæmorrhages from various textures, sloughing of the tonsils, breaking up and crenate form of blood-discs, cuticle separating under pressure before death, &c. Here there was no particle of hæmoptysis.

five cases (observed at Brompton, and at University College Hospital) of chronic bronchitis with or without marked emphysema (*but always without serious disease of the heart*), the absence of such expectoration was noted in nineteen cases, its presence in six. Now in *all* these six cases of streaked expectoration, there was more or less ground for suspicion that tubercles were to a slight amount present,—in two of them this was proved to be the fact by *post-mortem* examination. While, then, as I have found, bloody expectoration occurs in 71·79 per cent. of tuberculised persons in the first stage, it occurred in 24 per cent. of bronchitic people, free from serious cardiac disease; but in all of the latter there was either suspicion or certainty of the existence of tubercle to a *slight* amount: pathologically, these people were *latently* tuberculous persons, with super-added bronchitis; but practically, they could only be regarded as bronchitic. The mean duration of the disease in the phthisical cases was 26·55 months, in the bronchitic, 49·50, hence the significance (*quoad* tubercle) of hæmoptysis is greater even than the relative per-centages above given would signify. Streaked or tinged sputa are rarely or never the 'first symptom' of phthisis; should they appear in this guise, then, they would probably be dependent on some other cause. The question of hæmoptysis in plastic bronchitis has already been referred to [575].

890. "*Primary cancer of the lung and mediastina*, as I have elsewhere shown, from the analysis of a small number of recorded cases, is very frequently attended with sanguineous expectoration or pure hæmoptysis. In regard of this symptom, the two diseases may be thus compared:—the per-centage of hæmoptysis *of all amounts* in cases of cancer is 72, in phthisis 80·92; while hæmoptysis *above one ounce* occurs in cancer and phthisis in the ratio of about 70 to 40. Hence 100 cases of cancer of the lung will be attended nearly as often with hæmoptysis of all amounts, and greatly more often with hæmoptysis above an ounce in amount at a time, than 100 cases

of phthisis. But, on the other hand, tuberculous is so vastly more frequent than cancerous disease of the lung, that the share of the population suffering at any time from cancerous hæmoptysis will form but an insignificant fraction of that suffering from hæmoptysis of tuberculous origin."

891. The relationship of *empyema* to hæmoptysis has already [679] been considered.

892. "*Simple chronic consolidation of the lung* has not, in my experience, been attended with hæmoptysis to any amount.

893. "*Acute pneumonia*, accompanied with discharge of pure blood, is almost positively connected with tuberculous disease.

894. "*Gangrene* of the lung is rarely attended with hæmoptysis in the adult; in infancy (when tuberculous hæmoptysis is very rare) it is rather common.

895. "I have never known *ulceration of the larynx* productive of discharge of blood to any extent; streaks are not uncommon. But ulceration of the larynx, proceeding from within outwards, seems not to occur as a primary affection: at least I have never seen it except in follicular sore-throat, phthisis (which may be completely latent in regard of the lung), cancer, and syphilis.

896. "Hæmoptysis arising from *disease of the heart* can with difficulty be confounded, even in itself, with the severer forms of phthisical hæmorrhage; while the physical signs of the cardiac disease will point to its true source in such cases of the slighter form. I have never once seen cardiac disease, of such kind as to cause hæmoptysis, coexistent with *phthisis*, using the term in its practical sense; but in a fair number of instances I have seen advanced cardiac disease in persons whose lungs contained *crude tubercles and gray granulations.* It may be, therefore, that the conditions of the system existing in heart-disease are unfavourable to the development of tubercle; but the infrequency with which the two kinds of disease are found together doubtless depends, in the main, on the

difference in the periods of life at which each is especially prone to occur.

897. "*Aortic aneurism*, opening into the trachea, may (without proving immediately fatal) give rise to hæmorrhage, indistinguishable by its own characters from profuse pulmonary hæmorrhage. The history of the case, the physical signs, the age of the individual, &c., commonly establish the diagnosis; but when the aneurism is small, and so placed as to elude percussion, and pressure-signs, both concentric and eccentric, are absent, the difficulty of *proving* the existence of aneurism may be insurmountable; the existence of the disease may be divined, but not demonstrated. It is to be remembered that the absence of notable signs of tuberculisation does not justify the inference that the hæmoptysis is not phthisical, seeing that a tremendous pulmonary hæmorrhage may occur when slight consolidation exists at one apex only, and that such consolidation might be supposed to depend on the pressure of an aneurism.

898. "It is matter of common belief that in women who menstruate imperfectly and irregularly, the expectoration of a small quantity of blood is insignificant. I think this *perhaps* true where streaks only are concerned; but in every instance I have observed (*except one*), where such succedaneous hæmoptysis reached an ounce or upwards, there has been either evidence, or ground for suspicion, of tuberculisation. Similarly I have seen two cases of individuals presumedly in a state of perfect health, who, in a violent fit of passion brought up a certain quantity of blood from the lungs: *both had latent tubercles.*"*

899. I have met with several instances, where individuals, apparently in perfect health, have under violent effort had more or less copious hæmoptysis. In some of these cases rapidly destructive phthisis has ensued: but close inquiry has then invariably disclosed evidences of previous failure of health.

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* Author's Report on Consumption. Brit. and For. Med. Rev. Jan., 1849.

And again, there seems much reason to believe that where the ascent of mountains produces hæmoptysis, the lungs are already latently diseased. We cannot, otherwise, understand why so very small a proportion of those making such ascents, expectorate blood.*

900. The tendency of my experience, then, is clearly to show the vast frequency with which hæmoptysis is in some manner or other an attendant on tuberculous disease. The fact, that individuals are occasionally met with who, after having had more or less profuse hæmoptysis, live on to a good age without exhibiting phthisical symptoms, does not invalidate this result; it simply shows that tuberculisation tending to hæmoptysis may, as well as that not so tending, undergo spontaneous suspension.

901. Hæmoptoic blood from the lungs is in the great majority of cases brightly florid; but it may be of mixed arterial and venous tints; or it may be wholly dark, almost black. It is aërated and frothy, unless very copiously and rapidly ejected. When in moderate quantity the blood is voided by the act of expectoration; it escapes in gulps from the mouth when profuse; or may be ejected with efforts resembling those of vomiting.

902. The *diagnosis* of hæmoptysis in general may be considered in the present place; while we reserve for future consideration any special points connected with its distinction, in particular diseases of the lungs and heart.

903. Hæmorrhage from the mouth and fauces can be distinguished by careful inspection of these parts from hæmoptysis. Epistaxis, under ordinary circumstances, cannot be confounded with hæmoptysis; but sometimes blood, instead

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* Boussingault, d'Orbigny, and Roullin, make no reference to hæmoptysis, as having occurred in their ascents of the Andes; de Saussure observed nothing of the sort in his ascent of Mont Blanc; and Mr. Albert Smith (whose medical education gives value to his testimony) insisting in his narrative on the difficulty of breathing experienced, is silent concerning expectoration of blood, and yet his party and guides numbered twenty-four.

of coming forwards from the nares, trickles backwards, and may be from time to time coughed up. But here, again, close examination of the nares anteriorly, and of the pharynx, will disclose the source of the hæmorrhage.

904. Patients will often persist in assigning to the throat hæmorrhage, which in reality comes from the lungs, because they first become conscious of the presence of blood, when it reaches the former part: this is a source of fallacy against which the young practitioner must be on his guard.

905. Hæmoptysis is distinguished from hæmatemesis by the following characters.

Hæmoptysis is most frequent between the ages of eighteen and twenty-five; hæmatemesis, unless vicarious from defective menstruation, rare before the thirtieth year: to both these propositions there are however numerous exceptions. Sex is valueless as a guide.

The previous history in the one case points commonly to thoracic disease, in the other to gastric or other chylopoietic disturbance.

Hæmoptysis is immediately preceded by slight dyspnœa, anomalous sensations about the chest, tightness, weight behind the sternum, or at some other spot of the thorax, to which the patient will sometimes confidently point as the seat of mischief; hæmatemesis by weight and uneasiness at the epigastrium, sometimes by nausea. A salt taste in the mouth, with tickling and guggling sensations in the throat, often precedes actual hæmoptysis; whereas I certainly have never known this complained of in hæmatemesis.

Blood is ejected in severe hæmoptysis with efforts undisguishable by patients from those of true vomiting; but previously to such "vomiting of blood," mouthfuls have generally been coughed up: while in some cases of hæmatemesis the blood regurgitates, rather than is vomited, and nausea even is wanting. Hæmatemesis is attended by tenderness at the epigastrium; hæmoptysis by none of this. No matter what amount of blood be brought up from the lungs at once, small quantities continue to be expectorated for a time; when the stomach is at fault, on the contrary, full discharge occurs suddenly, and there is, generally speaking, an end of the matter,—certainly no bloody or stained sputa follow. In hæmoptysis the blood is florid and frothy; in hæmatemesis dark and non-aërated: at least this is the common case. But when large masses of blood are discharged from the lungs, they may be totally frothless: and where hæmorrhage occurs rapidly from an artery into the stomach, as in cases of simple chronic ulcer, the blood is vomited at once, and of perfectly arterial hue; no time is allowed for discoloration by the gastric fluids. On the other hand the blood of hæmoptysis may be partly, or wholly, of venous tint,—but, so far as I have seen, it never has the grumous pitch-like appearance of blood ejected from the stomach. The blood of hæmatemesis is sometimes of acid reaction; and the blood-discs altered in outline by the action of the gastric fluids: but when these evidences exist, the case will commonly on other grounds be a tolerably clear one. Discharge of blood by stool is the rule in hæmatemesis; the exception in hæmoptysis: in the latter case, it comes of blood accidentally swallowed, and is never, so far as I have known, abundant.* In hæmoptysis liquid rhonchus may almost invariably be found in some part of one or both lungs; nothing of the sort exists in hæmatemesis. When the lung supplies the blood, the pulse is oftener excited, full, bounding (sometimes *bisferiens*), than when the stomach is its source: in the former case, the pulse is proportionally less quickened than the respiration; still this perversion may occur in hæmatemesis also. Lastly, the diagnosis should never be fixed on without making a careful, yet cautious examination of the chest. Should the evidence of chronic changes at the apices exist, a doubtful opinion in favour of pulmonary origin would at once be strengthened. But the absence of such changes would not exclude the possibility of hæmoptysis; for, as will hereafter fully appear, such discharge of blood may occur before any notable physical changes have occurred in the lungs; nor, on the other hand, does latent tuberculisation exclude the possibility of hæmatemesis. The state of the chylopoietic viscera should be examined physically in aid of the diagnosis of hæmatemesis.

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* To this there is one exception. Patients, bleeding from the lungs, will sometimes continue steadily to swallow the blood, for the purpose of soothing the apprehensions of those about them; in a case of this kind I have seen really enormous quantities of blood in the alvine discharges.

906. The *treatment* of hæmoptysis, during its actual existence, aims (*a*) at removing the conditions causing it, or (*b*) at stopping it in spite of those conditions.

907. (*a*) Now if there be evidence of congestion of the lung of an active kind, with febrile excitement and strong cardiac action, that congestion should be treated by bleeding from the arm, to an amount measured by the urgency of the symptoms, and the constitution of the individual. Slow local bleeding from the chest by leeches I believe to be highly objectionable; the rapid abstraction of blood by cupping, however, if the patient be enfeebled by previous disease, is preferable to venesection. The head should be kept high during the bleeding; and, indeed, throughout the progress of the case; a semi-faint state tends in itself to control hæmorrhage. Leeches to the anus, or to the feet, followed by the hot pediluvium, sometimes very manifestly control hæmoptysis, where there are evidences of abdominal congestion. Nauseating doses of tartarised antimony, or of ipecacuanha, are, by some had recourse to from the first; but the practice is one of which I have little experience. Tartarised antimony, it is affirmed on high clinical authority, has actually caused death under these circumstances; however, it does not necessarily increase bleeding, even though it causes vomiting,—this I have *seen* in the practice of others. The bowels should be freely opened with

cooling saline purgatives, and watery evacuations if possible kept up for a day or two.

Ligature of the limbs, so as to prevent the free return of blood through the veins, has proved a timely adjuvant occasionally: Junod's exhausting apparatus will be useful on the same principle. Raising the arms over the head unquestionably stops epistaxis sometimes; I know not what effect the position may have in hæmoptysis. Free circulation of cool air, light bedclothes, a hard bed, quietude, and silence are essential aids. Large pieces of Wenham Lake ice should be allowed to dissolve in the mouth; and the *cautious* application of ice in bags to the spine or over the heart I have repeatedly seen (hence I do not value the speculative objections to the practice) almost instantaneously arrest the flow of blood. Heat may at the same time be applied to the extremities.

Among remedies, controlling the action of the heart, digitalis, aconite, and prussic acid claim attention; if the heart be irritable, and the hæmoptysis moderate, the first-mentioned medicine is valuable. Refrigerants, such as nitrate of potass, sulphuric and other acids, may be employed as adjuvants.

908. The medicines qualified to fulfil the second indication, are the acetate of lead, given in doses of two to four grains with dilute acetic acid and laudanum, every half-hour, hour, or two hours, according to the urgency of the case; alum; ergot of rye (not so valuable, however, as in epistaxis), matico, and gallic, acid. My recent experience leads me to place greater trust in gallic acid than in any other agent of the class. But if the hæmorrhage be at all severe, the doses must be large and very frequently repeated: twenty to thirty grains should be given every half-hour at first,—the dose and the frequency of repetition being gradually lessened. I have seen no ill effects from these large doses.* If there be excessive anæmia, the

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* The blood expectorated, while gallic acid is taken, often exhibits (as I first observed in a case seen with Dr. D. Fraser) a peculiar green tint, deep,

tincture of the sesqui-chloride of iron, or sulphate of iron with gallic acid (making a gallate of iron) may be given from the first. In various other astringents, krameria, logwood, kino, catechu, sulphates of zinc and copper, little trust is to be reposed. Nor, useful as it is in some hæmorrhages, have I often seen turpentine distinctly efficacious in hæmoptysis: still it sometimes appears decidedly beneficial. A provincial chemist told me some time since, that, on one occasion, when he had in a series of closely following attacks brought up "several pints" of blood, lead and all ordinary remedies failed, while turpentine, taken internally and rubbed into the chest and limbs, distinctly arrested the bleeding. Drachm doses of kitchen salt, either in powder or dissolved in water, appear sometimes (I have seen the fact in three instances) to arrest hæmoptysis very rapidly,—and this, whether they produce emesis or not. Five or six drachms may be given at intervals,—and, as the agent is always at hand, it may at once be used, while other means are in preparation.

909. The treatment of hæmorrhagic reaction and of sinking is the same in the case of hæmoptysis as of all active hæmorrhages.

910. Are there any means of preventing the tendency to frequent hæmoptysis in the course of tuberculous disease? I believe not. Cheyne, of Dublin, it is true, had great faith in the prophylactic virtues of small and repeated bleedings; but I confess that the case cited from his practice by Sir James Clark hardly makes me a convert. Hæmorrhage was clearly not prevented in this instance (though a *weekly* venesection was performed for a year), for the bleedings recurred again and again; and blood-letting seems eventually to have failed even to control the seizures, when actually present. I fully agree with Sir James Clark, that the remedy was relied on too exclusively; besides I cannot believe that this patient's case was the type of

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but transparent: a very evident proof that the acid passes through the circulation.

a class, for, instead of becoming spanæmic and emaciated, as the mass of men would under such treatment, he appears to have grown into flesh and regained strength.

ATROPHY OF THE LUNG.

911. The density of the lungs and the quantity of blood they admit, diminish with age; their structure undergoes throughout a normal atrophy.

912. Hourmann and Dechambre* describe three types of change in intimate structure. In the least advanced the air-cells are rounded and considerably enlarged: in the second stage, the cells elongated into ellipses, so as to look like chinks, are still further enlarged; while the lobules present the same elliptical outline: in the third, the air-cells, greatly enlarged, uneven in size and without distinct form, give the appearance of torn net-work to a dried section of the lung; the distinction into lobules is lost.

913. The loss of vesicular walls and of inter-vesicular substance diminishes the positive bulk of the lung: the thorax contracts in consequence. The extent of aërating surface undergoes great reduction also; but as the excess of blood gradually lessens, no dyspnœa occurs. The chest-walls sink in; and in some highly marked cases serosity accumulates in the pleura to fill the space which would otherwise be left empty, through the inability of the chest to follow beyond a certain point the dwindling and receding lungs.

914. Local atrophy forms an important secondary constituent of a variety of pulmonary diseases; and, according as diminution in the amount of circulating blood occurs or not, will obstructed breathing and subjective dyspnœa ensue, proportionally to the extent of wasted texture.

Thus, atrophy forms an essential element of vesicular emphysema. Local wasting of the parenchyma follows its infiltration with induration-matter, tubercle, or carcinoma. Mediastinal growths or aneurism, interfering with the supply of blood by pressure on the bronchial or pulmonary arteries, impede nutrition locally. Pressure on a main bronchus, if long continued, first causes collapse of the lung, and subsequently atrophy from inaction. Further, exudation-matter in the pleura sometimes tightly embraces a portion of lung, and causes its atrophy by obstruction of its vessels.

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* Arch. Gén. de Médicine, 1835.

HYPERTROPHY OF THE LUNG.

915. Hypertrophy of the lung, unless when accidentally ensuing upon emphysema, cannot be regarded in the light of a disease. It arises as the result of inaction of the fellow-organ, as when this has been compressed and rendered permanently useless by empyema.

916. The side may become considerably dilated; if the hyper-nutrition be limited to one lung, this is easily ascertained [680]. The percussion-sound grows clear, the vocal fremitus marked, the vocal resonance strong, and the respiratory sounds exaggerated and harsh, with an undue amount of expiration. Emphysematous distension of the air-cells sometimes advances *pari passu* with hypertrophy.

VESICULAR EMPHYSEMA.

917. The disease, inconveniently termed vesicular emphysema by Laennec (and which it might be better, perhaps, to call *rarefaction of the lung*), is essentially characterised by enlargement with attenuation of the air-cells, obliteration of their vessels, and atrophy of their walls. Occasionally oil, as first pointed out by Mr. Rainey, is discoverable in the walls of the vesicles, but it is not constant, and when present, its relationship, of cause or of effect, to the existing atrophy is uncertain. A state of hypertrophy of the inter-vesicular structure occurs in very rare instances.

Rarefaction, distension, and loss of elasticity are the most striking physical characters, practically considered, of emphysematous lungs. General distension is shown by the downward encroachment of the organs; by their overlapping in front at, and above, the level of the second costal cartilages; by their bearing on the surface more or less deep impressions of the ribs; * and by the encroachment of the left lung on the superficial cardiac region. Instead of the natural area of that region [373], we may have one the shape of a narrow isosceles triangle, the base measuring one third of an inch, the two vertical equal sides about two inches.* Local distension, again, is exhibited in irregular lobulated elevations of the surface by so-called subpleural sacculi;—sacculi formed by enormous distension of some air-cells and total destruction of others: they are in truth *air-cavities* beneath the pleura, or within the area of the lung.

918. Emphysema affects one or both lungs, and the entire, or, as is much more common, a part only of each organ. The upper division of the right, the lower of the left, seems to suffer most frequently.† The disease may be limited to some scattered vesicles, affect entire lobules or even lobes: in the latter case the interlobular cellular tissue disappears.‡ The surface of the organ suffers most; the entire substance being rarely affected, though deep-seated islets of the disease are not uncommon. The anterior edges seem particularly prone to emphysema; the subpleural sacculi are chiefly met with at the posterior bases and mediastinal surfaces.

919. The bronchi, commonly the seat of chronic inflammation, are sometimes dilated,—but not with sufficient frequency to suggest a relationship of cause and effect. Pleuritic adhesions are scarcely more common, pulmonary tubercle less common,§ than in persons of the same age cut off by all varieties of disease

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* Skinner, U. C. H., Males, vol. ix. p. 97.

† Louis, Mém. de la Soc. Méd. d'Observation, t. i.

‡ Lombard, de l'Emphysème pulmonaire, p. 8.

§ Louis, loc cit.

indiscriminately. Not only then is emphysema not a cause of tuberculisation, but the two diseases seem to a certain extent indisposed to alliance: they are not incompatible, it appears to me, on account of any special crases of the blood, as has been fancifully suggested; but simply seldom associated, because they are essentially diseases of different periods of life. The two affections are, in truth, sometimes found in activity in the same individual,—tubercle forming and softening although the so-called venosity of the blood is carried to the highest point. I believe, however, that as emphysema entails obliteration and destruction of minute vessels, it must within certain limits prevent the deposition of tubercle, as of any other morbid product: the rarity of pneumonia in highly emphysematous patients is indubitable. Local tuberculisation, again, as long since shown by Sir R. Carswell, may, by obstructing some air-cells, lead to the distension of adjoining ones. Interlobular emphysema, as also mediastinal and sub-cutaneous infiltration of air, are very rare sequences of the vesicular disease. A sub-pleural sacculus has been known to give way and produce pneumothorax.

920. Emphysema tends in the course of years to produce dilated hypertrophy of the right heart; and maintains more or less habitual venous congestion within the head, scarcely, however, demonstrably productive of either hæmorrhage or softening. What part, if any, it may play in generating Bright's disease seems to me as yet undetermined.

921. Pulmonary emphysema is essentially a disease of the chronic class. M. Louis, however, records a case where, at least symptomatically, it grew up in twenty-nine days. I have seen it, accompanied with interlobular emphysema, highly developed in an infant born without dyspnœa, and dying two hours after with obstructed breathing [879]. The freedom of respiration at birth opposes the idea of the disease having, in this instance, been congenital.

922. Dr. Jackson, junr., of Boston, U. S., found, that of twenty-eight emphysematous persons, eighteen had either a

father, a mother, or both, similarly affected; whereas, of fifty non-emphysematous people, three only sprang from emphysematous parents. Further, fourteen persons, emphysematous from early youth, came all of them of emphysematous stock; whereas, of fourteen, first affected at an advanced period of life, two only were of emphysematous parentage. These facts prove emphysema to be much more strongly hereditary than phthisis.

Symptoms and Signs.

923. The sole symptom of vesicular emphysema *per se* is dyspnœa. Often commencing in early youth, or even infancy, at first slight in amount, and only felt on some unusual exertion, such as running up-stairs; when once developed, dyspnœa is permanent, but subject to great variations in intensity. The patient feels as if his chest were never thoroughly, at least naturally, emptied of its air; and is conscious of an annoying sense of inflation or distension. On the other hand, it is true, many persons labouring under emphysema will affirm that their dyspnœa is only occasional,—that habitually their respiration is perfectly natural. But I have never known an instance of this kind where the patient was not the victim of a delusion; the truth was, that a moderate amount of dyspnœa had become to him second nature, a state of comfort and health,—and excessive difficulty of breathing alone gave him annoyance. The dyspnœa is increased, from time to time, through bronchial spasm, through abdominal infarction, or through intercurrent bronchitis: the former two causes produce sudden paroxysmal attacks, requiring the patient to rush to the open window for air, or pass the night in the sitting posture out of bed; the latter is less violent, but more protracted, in its action. The amount of persistent dyspnœa is generally proportional to the duration of the disease. The paroxysm occasionally induces a fit of palpitation, but not of any severity, unless some cardiac disease co-exist. Cough preceding, commencing with, or

following the first establishment of dyspnœa, exists in almost every case; but may be wholly wanting. The habitual sputa are frothy, liquid, and mucous or watery. It is matter of doubt whether the state of the lung itself, independently of that of the bronchi and pleura, may give rise to pain; my own observation leads me to doubt it. The facies of emphysematous patients is peculiar: of dusky colour, and anxious melancholy expression, it is full, out of proportion with the chest and body generally,—a probable result of thickening of the cellular membrane and muscles of the face, as suggested by Dr. Stokes,—the former from repeated venous obstruction, the latter from respiratory effort. The nostrils are thick, and very often the lower lip full, and venously turgid; I have, however, not seen the latter state, carried to extremes, without heart-disease. The muscles of the neck enlarge, and its cellular tissue disappears. The patient's gait is stooping; Dr. Stokes has known the acromial, interscapular and lower scapular regions almost horizontal. The strength is inversely as the dyspnœa; in aggravated cases, bodily exertion becomes an impossibility. The fat and flesh of the body generally and slowly waste,—each attack of bronchitis diminishing the weight *pro tempore*, or permanently. The pulse is not accelerated,—far from this, it ranges below the average of health in a considerable number of cases, except when intercurrent bronchitis is present. The respiration also, with the same qualification, is less frequent than in health; in fact, the act is so laboured a one, that it cannot be often repeated in the minute. The pulse often strikes the observer by its weakness, as compared with the amount of cardiac impulse. This want of accordance comes of the frequency with which the right heart undergoes enlargement, whereas, as a consequence of the pulmonary disease, the left does not suffer. No conceivable amount of emphysema, even of both lungs, will necessarily entail the smallest amount of dropsy,—even œdema of the ankles. If dropsy occur, there is something else (generally tricuspid regurgitation) to account for it. The bowels are habitually constipated; the urine aqueous.

924. The physical signs of emphysema are numerous and positive. Inspection discovers bulging of the infra-clavicular, mammary, and central sternal regions, or of the anterior surface, uniformly. General expansion of the chest occurs very rarely; M. Louis observed it only once in ninety-six cases: when it exists, it gives the thorax a globular form.

The state of the interspaces in the bulged portions of surface has been matter of dispute. Dr. Stokes has taught that in emphysema, "even after great dilatation of the chest has occurred, we see the intercostal spaces, so far from being obliterated, *deeply marked;*" and that the single malady in which this obliteration really occurs is pleurisy in its advanced stages. The conditions directly conducive to its production are *paralysis* of the intercostal muscles and centrifugal pressure, one being as essential as the other; this paralysis is presumed to be the result of inflammation extending to the muscular tissue. For the same reason, the intercostal spaces will not be obliterated in cases of simple hydrothorax, nor in *all* instances of pleuritic effusion; because muscular inflammation and paralysis do not exist at all in the former, and are not *necessarily* present in the latter. The question here started is strictly one of observation; and it must be confessed that the experience of physicians generally does not accord with that of Dr. Stokes in respect of the bulging of emphysema. MM. Louis and Woillez are wholly opposed to Dr. Stokes on this point. Both maintain that the intercostal hollows are in this affection either effaced or manifestly less marked than in the natural state; and even point out this implication of the muscular plane of these spaces as one of the distinctive marks of emphysematous prominence. It seems, however, possible to reconcile these conflicting opinions. I believe, in point of fact, that in emphysema the conditions in respect of bulging are of three distinct kinds. *First,* there may be no expansion at all, general or local;

secondly, there may be bulging of the surface generally, with a natural state of the intercostal spaces; and *thirdly*, there may be bulging with distinct obliteration of the intercostal hollows. The key to these apparent contradictions lies mainly in the anatomy of the disease—in its anatomical varieties, which have in this point of view escaped the consideration of the observers referred to. In the *first case*, I have found the disease, which, in respect of symptoms, may have been very intensely marked, of the *atrophous* kind, with but *slight* distension of the lung; here the physical cause of expansion was altogether wanting. The *second variety* of prominence I have never observed in the infra-clavicular region (when alteration of shape was limited to that region, one of the special seats of such change in emphysema), but have met with it elsewhere in certain cases of almost globular expansion of the thorax in emaciated emphysematous persons. But in these instances—and doubtless they exist more frequently than they are discovered or suspected—the local prominence, when characterised in the manner now referred to, was in all probability, especially when occurring at the back, a natural conformation, and wholly *independent of the emphysema*. *Thirdly*, when bulging has existed in regions where observation proves it to appertain specially to emphysema, such as the infra-clavicular, I have found the intercostal spaces distinctly prominent, and the disease, if opportunity for *post-mortem* inquiry presented itself, either of the *hypertrophous* kind, or of the atrophous variety, with great distension of the lung and sub-pleural sacculi.*

The inspiratory motion of expansion is greatly diminished;

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* The difference of opinion under consideration appears to be very readily explicable in the manner now proposed. What I have stated I believe to be in strict conformity with observation; whereas I am not aware that Dr. Stokes' theory, of inflammation of the intercostal muscles being a necessary condition of their eccentric displacement, rests upon any observed cases, submitted to close anatomical examination. It is not easy to understand, in his theory, why the intercostal muscles should resist eccentric pressure more powerfully than the ribs; and this, be it noted, equally in frames of every degree of

the lower part of the sternum and adjoining cartilages, in aggravated cases, even sinking inwards during inspiration,—nay, the base of the chest generally may then slightly retract. On the other hand, the elevation-motions are exaggerated,—the whole chest is raised, with the shoulders, as one piece. The duration of the expiratory movement considerably exceeds that of the inspiratory; both acts are laborious,—the expiratory, because the elasticity of the lung is destroyed by the disease,—the inspiratory, because the previous expiration has failed to empty the lungs to the normal amount, and the chest is with difficulty further dilated. The faculty for prolongation of the expiratory act, is probably derived from hypertrophy of the muscular coat of the bronchial tubes;—their expressing power is permanently taxed to the utmost. The respiratory action of the intercostal planes may be completely perverted,—bulging occurring in inspiration; the latero-inferior interspaces may bulge outwards beyond the level of the ribs in expiration.*

925. The condition of vocal fremitus varies; it may fall below, equal or exceed the average of health.

926. The semi-circular measurement of one side, or of the whole chest, is increased; in right-handed emphysematous persons, as first noticed by M. Woillez, the excess of width of the right over the left half of the chest inferiorly ranges lower than natural,—showing by inference the greater frequency of emphysema of the lower part of the left lung. The disease may, however, be highly marked without any such increase.

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muscular weakness or vigour. It appears to me that there will always be more or less hollow in the intercostal spaces, as has been shown by M. Woillez, so long as the elasticity or concentric force of the lung is not destroyed; that as soon as this change has taken place, as, for example, from the progress of emphysema, pressure sets in and influences the position of the intercostal muscles at least as readily as that of the ribs.

* The permanent lung-distension increases the buoyancy of the trunk: an emphysematous person once remarked to me that, though his dyspnœa grew worse, his power of swimming improved.

927. The percussion-sound is greatly clearer than natural, of exaggerated pulmonary, more or less tympanitic, quality, while the resilience of the chest-walls is augmented. I have never found the parietal distension sufficient to deaden the sound [140]. The morbid clearness extends beyond the middle line, if one lung only be affected. The natural resonance at the sternal notch, where no lung exists in health, is exchanged for emphysematous clearness; this fact, which depends on the junction of the edges of the lungs taking place higher than natural, in consequence of their distension, is very easily ascertained, both in the living and dead. Forced expiration has little or no effect in diminishing the amount, or contracting the area, of the morbidly clear resonance.

928. The respiration in emphysema belongs to one or other of two main types; it is weak, or of raised pitch, and altered quality. When of weak type, the failure may amount to actual suppression in some points; while in others, exaggerated respiration of harsh quality exists. Of the occasional existence of such exaggeration in lung-substance, on the confines of circumscribed emphysema, I have satisfied myself by a considerable number of observations; but, on the whole it is rare. Respiration of raised pitch, loud, harsh, and uneven, exists, in many cases, immediately over emphysema carried anatomically to the very highest point; the quality is sharply sibilant, and in part it may be constituted by a sibilant rhonchus in the very finest tubes.

The rhythm of the sounds is variously altered. Inspiration may be deferred [183]; and the two sounds both jerking and divided [182]. The inspiratory sound is comparatively short, the expiratory greatly prolonged: the ratio of the former to the latter sound may be changed from 3 : 1 to 1 : 4; in other words, the latter sound made *twelve* times longer in proportion to the former than in health. In such cases of enormous prolongation of expiration, this is rather a fine sibilant rhonchus than true respiratory sound. Rhonchi, bronchial, and dry or moist, are evidences of bronchitis, or of spasm of the tubes: very commonly, when an emphysematous patient seeks advice both bases are the seat of fine bubbling rhonchus. None of these rhonchal sounds owe their existence directly to emphysema: a peculiar dry crackling noise, by some termed a rhonchus, of doubtful mechanism, and rarely heard [214] forms, along with dry grazing friction-sound produced by prominent air-distended nodules, the sole direct rhonchoid sign of the disease.

The vocal resonance varies. It may be normal, null or bronchophonic. When very feeble, this apparently depends on weakness of the laryngeal voice,—itself caused in turn by the imperfect supply of air and laboured process of expiration.

The heart's sounds are feebly transmitted through emphysematous texture.

929. Signs of importance are derived from the heart. If one lung only be affected, the heart is pushed slightly to the opposite side; if both are implicated, downwards, and commonly to the right somewhat, inasmuch as the left lung is generally more affected than the right, at the base. The præcordial region, filled with distended lung, is bulged forwards, and sounds clear under percussion; no cardiac impulse may be felt within its area,—that impulse being transferred to the epigastrium. Enlargement of the right side of the heart, however, plays its part in thus altering the seat of impulse. The jugular veins may be swollen, though the heart is not obviously diseased; but I have never known them pulsatile, without co-existent affection of that organ. Emphysema is sometimes accompanied with visible arterial pulse: but I have not seen this, unless the patient were aged, or the subject of aortic regurgitation.

30. The only affection with which emphysema can be physically confounded is pneumothorax. The distinction is not difficult. In emphysema the percussion is less clear, less truly tympanitic than in pneumothorax; the side is less dilated; there

is more respiration (and, possibly, loud, sibilant, respiration, as we have seen), and such respiration as exists is superficial, instead of being deep-seated and distant, as in pneumothorax. Besides, pneumothorax affects only one side (double pneumothorax must be almost instantly fatal); whereas if emphysema exist to such a degree as to simulate in regard of percussion-sound the presence of air in the pleura, both lungs prove to be very seriously affected. Lastly, the symptoms of pneumothorax come on suddenly, and are greatly more urgent than those of emphysema.

931. The anatomy of emphysema prepares us to fear that its radical cure is impossible; we have no means of causing reproduction of destroyed lung. Experience, unfortunately, goes farther than this, and shows that *permanent* palliation even is most difficult of accomplishment; *temporary* relief is, on the contrary, most readily effected; the fair aim of art is, then, to ascertain, how a state of brief ease may be indefinitely prolonged.

The proofs that great amelioration is actually produced by treatment, are furnished not only by favourable change in the symptoms and in the patient's feelings, but by changes in the physical signs: the morbid clearness of percussion may be lessened, the quantum of respiration increased, and the distension of the lung reduced; a fact positively demonstrable by increase in the area of the heart's superficial dulness.* But what organic change in the lung-substance do these alterations in physical signs demonstrate? Not assuredly, that atrophy is gone, but that *distension* is reduced. And the reduction of distension is effected by the removal of bronchitis and the relaxation of spasm.

932. In nine cases out of ten, when a sufferer from emphysema applies for relief, sub-acute, rarely very acute, bronchitis is present. Local bleeding, blistering, and small doses of tartar

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* *Vide* case of Hope, Clin. Lect., loc. cit. April, 1849.

emetic are the essential agents for its removal. The other remedies, spoken of under the head of bronchitis, are not to be forgotten, however, when the first vigour of the inter-current disease is controlled. When emphysema exists to any amount, caution in the employment of depletory measures is essentially called for.

933. For the habitual chronic bronchitic state, I know of no combination superior to the æthereal tincture of lobelia inflata, with ipecacuanha, in ammoniacum mixture. Fits of dyspnœa are to be relieved by extract of cannabis indica in half-grain to grain doses, or by belladonna and stramonium, either in the form of pill or smoked in cigarettes. Stramonium alone may be used in an ordinary pipe, and may be more safely entrusted to patients than belladonna: it may be smoked until the head begins to ache slightly. Opium, however, must be had recourse to in very severe attacks, and may be pushed until sensible, but slight narcotic affects are produced. The camphor cigarettes of M. Raspail occasionally afford much relief.

934. Transmission of a gentle galvanic current from the nucha to the epigastrium, will sometimes avert, almost invariably mitigate and postpone, the paroxysm of dyspnœa.* An emetic (sulphate of zinc) will do so also: especially when the attack is connected with a loaded state of stomach. Cajuput oil and other carminatives, with soda, put a term to that caused by flatulent distension. The statements made concerning chloroform in the treatment of simple spasmodic asthma [621] hold true only to a certain extent of asthmatic seizures accompanying emphysema: that is, relief in the paroxysm is here easily obtainable; but on the other hand serious and permanent impression on the malady cannot be even hoped for.

Strychnia has been recommended for the purpose of improving the tone of the non-striated fibre of the bronchial tubes. I have tried it in a small number of cases, both endermically and by the mouth, in sufficient doses to produce obvious effects on the voluntary muscles, without in the slightest degree modifying the symptoms of the emphysema.

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* M. Henley, Males, U. C. H., vol. vi. p. 126, April, 1851.

935. Regularity of habits, and moderation in indulgence at table, are essential to the well-being of emphysematous sufferers; and it is obvious, from their forced obedience in regard of these points, that their lives are often (a questionable boon, it may be, as they themselves have more than once said to me) prolonged beyond the average term of existence.

936. Change of air is singularly beneficial, or singularly detrimental, according to the nature of the change; but no amount of experience will enable the physician to predict positively what manner of air, soil, or geological formation will best suit any individual case.

INTERLOBULAR EMPHYSEMA.

937. In interlobular emphysema air accumulates in the cellular tissue between the lobules. Very seldom associated with vesicular emphysema, this is an acute affection produced by sudden rupture of air-cells, leads to infiltration of air into the mediastina, and lays the ground-work of one variety of external sub-cutaneous emphysema.

938. Interlobular emphysema is commonly produced by violent efforts, which require the abrupt introduction of a large quantity of air into the lungs, and its forcible retention therein by closure of the glottis. The efforts in parturition, defecation, raising weights, coitus, violent coughing, paroxysms of rage, excessive laughter, and hysterical convulsion, have all been followed by rupture of the air-cells, interlobular, and occasionally subcutaneous, emphysema.

939. Carried to extremes interlobular emphysema may cause sudden death.* Martini enumerates forcible lung-insufflation among the modes of perpetrating infanticide. The experiments

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* Cyclopædia of Surgery, art. Emphysema, p. 79.

of Leroy d'Etiolles on animals show that death may thus be produced as suddenly as by dividing the medulla oblongata.

940. Are there any special signs of interlobular emphysema? I know of none. It is quite possible, as Laennec originally taught, that, if the surfaces of the interlobular spaces become prominent through distention, grazing, dry, friction-sound may be heard; but there will be nothing distinguishing the sound thus produced from that caused by the sub-pleural sacculi of advanced vesicular emphysema. The same observations apply to Laennec's "dry, crepitant rhonchus, with large bubbles."

ADVENTITIOUS PRODUCTS.

941. The lungs give origin to a variety of Adventitious Products: among Precipitates, to Saline Concretions and Fat; among Deposits to Typhoid, Tuberculous, Syphilitic, Melanic, and Purulent matter; among Growths to Hæmatoma, Fibroma, Osteoma and Carcinoma; among Pseudo-Tissues to Induration Matter; among Parasites to Acephalocysts and certain Entophytes.

942. Few of these products, however, require separate consideration in a practical work. Saline concretions, fat, melanic and purulent matter, and induration-matter are met with only as attendants on various morbid states, clinically more important than themselves. Certain others of the products enumerated are of no practical significance; and we shall confine ourselves to the consideration of typhoid, tuberculous and syphilitic deposits, carcinoma, and parasites.

TYPHOID DEPOSIT.

943. The lung-substance, as well as the trachæal and bronchial walls, becomes in certain cases of typhoid fever the seat of the non-plastic typhoid deposit, which accumulates under the patches of Peyer and in the mesenteric glands. On the other hand, as

long since taught by M. Louis, young persons convalescing imperfectly from typhoid fever not unoften fall into pulmonary consumption of rapidly fatal course.

944. That death does occur in some cases, after typhoid fever, with rapid breaking up of the tissue of the lungs, and many of the symptoms of consumption, is indubitable. But it has always been a matter of difficulty to explain the connection between typhoid fever and tuberculisation of the lungs. M. Louis, referring the latter to the prolonged febrile action of the former, can scarcely be said to have settled the point. Is it possible that unusually abundant deposition of typhoid matter may in these cases be the real groundwork of the rapid destruction of the lungs? The question, beset with difficulties, it is true, seems worth investigating.

TUBERCLE.

945. Tubercle in the lung constitutes the anatomical character of phthisis, or consumption,—a disease which, in the vast majority of cases, runs a chronic, but occasionally an acute course.

CHRONIC PULMONARY CONSUMPTION.

946. The anatomical changes occurring in the lung in *progressive* chronic phthisis are referrible to three main stages; and these stages sometimes correspond to certain varieties in the symptoms, and always to modifications in the physical signs. There are, then, clinical as well as anatomical reasons for recognising three stages in the advancing disease, those of deposition and induration, of softening, and of excavation,—stages so well understood that the detailed account of them seems uncalled for in a practical work. In *retrogressive* chronic phthisis the anatomical conditions, being less satisfactorily established, will be by and by considered.

947. *Physical signs.*—The *first stage*,—that of accumulation of groups of gray granulations or crude tubercles in variable

quantity, and with or without intervening simple induration of tissue,—has the following physical signs.

948. The infra and supra-clavicular regions on the affected side (granting that one side only is affected) are either unaltered in form, or they are slightly flattened. The former is the more common of the two cases: probably, the diminished bulk of the apex, produced by deposition of tubercle and closure of *some* air-cells, is at first counterbalanced by the distension of *others*. It falls within the limits of the probable, indeed, that cases may now and then be met with, in which such emphysematous distension shall so predominate as to produce slight bulging over tuberculised lung. Many years ago, Dr. Chambers, without offering any explanation of the circumstance, mentioned to me that he had observed an increase in the antero-posterior diameter of the apex at the very outset of the disease. I have carefully watched for examples of the fact, but have never met with one, unless where the sound was clear under percussion, and the existence of local emphysema, therefore, strongly probable. It would appear, as matter of experience, to be impossible that tubercle alone, accumulated to such an amount as to impair in the least degree the clearness of percussion-sound, shall be the cause of bulging. The precise degree of tuberculisation, which will produce flattening, varies with many accidental circumstances, such as the relationship of the tubercles to the minuter bronchial tubes (if many of these be obstructed, local collapse of lung-substance will ensue), and the presence or absence of plastic contractile exudation within the parenchyma or on the pleural surface. Atrophy of the tissue of the lung can scarcely occur at the very earliest stage of bronchial obstruction. Flattening may sometimes very positively exist, but, from being equally shared by both sides, escape detection: when present to any extent, the clavicle is thrown into unnatural relief.

949. Vocal vibration is certainly increased in intensity under the clavicle; but the amount of increase is trifling. Its existence

is, therefore, with difficulty established under the right clavicle, where there is a natural excess of fremitus; and on the left side, as a sign of incipient tuberculisation, increased fremitus commonly fails us, because priority of disease on that side is most common in females, who, unfortunately, from the peculiarities of their voices, have naturally little or no vocal vibration.

950. Careful admeasurement with the callipers will sometimes detect very slight reduction in antero-posterior diameter at this period. If the deficiency be marked, pleural false membrane is probably present to some amount. The motions of the infra-clavicular region are perverted, to the eye, to the hand, and to the measure. The hand laid flat on the surface, instead of being arched outwards during inspiration, is simply raised upwards; elevation-movement exists, expansion-movement is wanting; nay, more, the infra-clavicular region may actually sink in with inspiration. The ribs, if the pleuræ are agglutinated, may be felt to converge at the same time. The deficiency of expansion-movement under the clavicle is a more valuable sign in the female than in the male, for the obvious reason that it is naturally much more limited in the latter than in the former.

951. The results of percussion may or may not be significant. Slightly impaired clearness of sound, and slightly increased parietal resistance, may be produced by a very few scattered tubercles at the apex,—local collapses of the lung doubtless contributing to impair the resonance. The deficiency will be earliest caught at the inner aspect of the supra-clavicular and clavicular regions (see Diagrams I. and II.), where the apex of the lung lies. To detect it, percussion must be very lightly made, and, for purposes of certainty, repeated in various postures both of the patient and of the percussor. Care must be taken to direct the percussion from, and not towards, the trachea: with this caution, percussion is much more conclusive at the inner than the outer part of the supra-clavicular region. Slight dulness is a more valuable sign in the female than in the male, and more valuable in both sexes at the left than the right side [115]. Habitually, a

greater superficial area of disease is required to affect the percussion-sound in the infra-clavicular and supra-scapular regions; but occasionally the latter suffers even before the front of the chest. Neither in front, nor behind, does the dulness extend beyond the middle line. Sometimes, when tubercles are sparingly scattered through an entire apex, percussion on a broad surface will disclose a difference in resonance, imperceptible when a single finger is used as the pleximeter. Or recourse may be had to the dynamic tests: the increase of clearness produced by a full inspiration will be, in comparison with that on the healthy side, very trifling; and, on the other hand, there will be comparatively a great diminution of clearness at the close of a complete expiration. As deposition, collapse, and consolidation advance, the sound becomes duller and duller, but never becomes totally toneless, or of putty-like flatness. If consolidation extend across from the costal surface to the trachea, or large bronchi, more especially if the pleural laminæ be agglutinated at the spot, the percussion-sound may be wooden, or even tubular. The fallacy of co-existent emphysema is always more or less to be apprehended during this stage.

952. The respiration in the infra-clavicular region, and also usually in the upper scapular, is affected in *intensity*, being weak and almost suppressed in some points, exaggerated in others; perverted in *rhythm*, being frequently jerking; impaired in *quality*, being harsh, bronchial, or even slightly blowing. The value of these states of respiration is directly as the limitation of the area within which they are discernible; if they exist above, and are imperceptible below, the second interspace, they are very seriously significant. Slight harshness of respiration is more valuable, as a sign, in the male than in the female, and notably so on the left than on the right side. If the other causes of jerking rhythm [181] can be excluded, which may or may not be difficult, this condition of rhythm, when limited to one apex (it is rarer posteriorly than anteriorly) becomes a really important sign of tuberculisation. My opinion

on this point has yearly grown more positive. It is to be remembered that it occurs at a period of the disease when the physical signs generally are few in number, not so decisive as might be wished, and when, of course, every addition to their number is really important. According to my experience, it is a persistent phenomenon. I have not, as Dr. Stokes appears to have done, succeeded in removing it by local treatment, even in cases where other signs, for instance, harshness of respiration, were favorably modified by that treatment. It may exist in the highest degree without any co-existent affection of the pleura; and must not be confounded with grazing friction-sound. Prolonged expiration, if unattended with alteration of quality, is insignificant: under such conditions, it is in all probability a normal state; and, even coupled with slight harshness and coarseness of quality, it must be cautiously received as evidence in females, and at the right side.

The only rhonchal sound specially belonging to this stage of phthisis is the dry crackling; occurring towards the close, it gradually passes, with the exceptions already referred to, into the humid crackling variety. Occasionally at the close of this stage the peculiar condition, I have designated as *cogged-wheel rhythm* of respiration, exists,—oftener in the supra-spinata fossa, than below the clavicle [186].

953. The vocal resonance varies to such an extent in amount and quality, as to make it totally unworthy of clinical confidence: I have known it (where the existence of consolidation was positive, either from other signs, or, in addition, from *post-mortem* examination) of the average characters of health, weak, null, exaggerated, intensely bronchophonic, or pectoriloquous. The state of vocal fremitus, curiously enough, does not vary thus, and is hence, comparatively, a more trustworthy guide: but its positive value, we have seen, is next to nothing. The reasons, why vocal resonance should thus vary, are amply explained in the First Part of this work.

954. In a doubtful case of tuberculisation of the right apex,

if the heart's sounds, but especially the first, be more clearly audible near the clavicle on that side than the left, we have herein presumptive proof of consolidation; but the absence of this sign will not disprove the existence of solidity, of which strong evidence appears on other grounds. Subclavian murmur may be present; and also systolic murmur at the second left, or pulmonary, cartilage, associated or not with basic systolic, or with subclavian, murmur. To the arterial pulmonary murmur, Dr. Latham attaches much importance, as an attendant on tuberculisation,—but I confess its diagnostic claims do not appear to me to be established: as to its frequent absence, I take it for granted that so positive a fact must be admitted on all hands.

955. The signs of dry or plastic pleurisy, and of bronchitis and of pneumonia, may occur in connection with tuberculous depositions; they have indirect value, if limited to one or both apices. Tuberculous patients, the *apex* of whose lung is the seat of capillary bronchitis, are of course liable to be seized with idiopathic bronchial inflammation of both *bases* from accidental causes. Under such circumstances, it is curious and interesting to observe the manner in which the upper and lower rhonchi travel towards each other so as eventually, in some rare instances, actually to meet towards the middle height of the lung. Where such union of the rhonchi takes place, the case is of the most serious character. I may further observe, with respect to the symptomatic rhonchi occurring in connection with tubercle at the apices of the lungs, that I have found true crepitation, to say the least, singularly rare: that is, unless in cases where the cause of the rhonchus is really extensive pneumonia,—such pneumonia as shall during its existence assume, in point of importance, all the characters of the idiopathic inflammation. Under the ordinary circumstances of acute irritation setting up in the neighbourhood of the new matter, the subcrepitant is the rhonchus audible, and capillary bronchitis the anatomical state present.

956. The signs of the *second stage*, that of softening, are in part new, in part those of the first stage, either stationary or carried to a higher point. To begin with the latter class: depression, both above and below the clavicle, is now greatly more marked, and may sometimes be really present to a notable amount, but be masked by twisting of the clavicle downwards and inwards on its long axis. When the clavicle thus, as it were, follows the retreating ribs, the callipers, or chest-measurer, supply the only trust-worthy evidence as to the amount of depression. The corresponding supra-scapular region, if one apex be more affected than the other, is distinctly hollower than its fellow,—a fact apparently so strange, that nothing but repeated observation would justify its statement. The vocal vibration does not increase as a consequence of the softening process; but as this is, generally speaking, accompanied with extension of induration also, such increase may occur. The semi-circular measurement of the side, and the transverse diameter of the chest in the axillæ, are lessened in consequence of the general deposition of tuberculous matter, atrophy, and interstitial contraction of the lung, together with, in some cases, contraction of pleural false membrane. Dulness under percussion gains in area and in intensity, and is, oftener than in the first stage, wooden or tubular. The respiration grows more extensively and markedly blowing, or remains merely bronchial. Vocal resonance varies as before.

957. The *new* phenomena in this stage are humid crackling, and thin metallic bubbling rhonchus. When elimination of the softened material commences, the rhonchus may become cavernous on a small scale.

In consequence of the diminution of the mass of the lung, the heart may be elevated above its natural position, the diaphragm raised, and the mediastinum drawn towards the mainly affected side. The heart's bulk gradually diminishes; but its area of superficial dulness may be apparently increased, in consequence of contraction of either, but especially of the left, lung.

958. The signs derived from inspection in the *third or excavation stage* remain as previously. I have, however, in some rare instances, known extreme infra-clavicular depression existing in the second stage diminish somewhat, nay, even give place to slight bulging, when a *capacious* excavation had formed. This will probably occur where the anterior wall of the excavation is excessively thin, indeed merely membranous; the condition becomes one, physically speaking, closely assimilable to local pneumothorax: at all events, of the fact I am positive. Again, in such cases, inspiratory expansion may improve to a certain extent. I have observed this in a case where amphoric respiration and cracked-metal percussion-sound gave evidence of the size of the excavation; and the more solidified the rest of the lung, the greater will be the expansion over the cavity.* Rhonchal fluctuation may sometimes be detected; and if the cavity be of large dimensions, fluctuation, produced by succussion of the trunk, may be felt.

959. The percussion-results depend less on the fact of cavity existing than on the conditions of the cavity. If it be small, and surrounded with much indurated lung, the sound will be absolutely dull, or wooden, or tubular. If there chance to be a thick stratum of sound tissue between the excavation and the ribs, gentle percussion may be of almost healthy quality,—moderate dulness, slightly tubular, too, is detected on strong percussion. If there be several small excavations, with indurated substance between them, the sound is markedly dull, and somewhat tubular; if one or two large excavations, amphoric or of cracked-metal quality.

960. The respiration, provided the cavity be neither distant from the surface, nor separated from it by a stratum of healthy tissue, is divided, hollow, hoarse, blowing, and cavernous, or actually amphoric; and this state alternates or co-exists, in the

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* Green, U. C. H., Females, vol. v., p. 148, July, 1850. Expansion is very rarely totally deficient over a large-sized cavity; greatly more rarely than over highly consolidated texture.

manner already described [194] with gurgling rhonchus. The cough is cavernous or amphoric; and metallic echo or metallic tinkling in some rare cases accompanies the voice, cough, and respiration. The vocal resonance varies in characters; it may be pectoriloquous, amphoric, bronchophonic, natural, weak, or null. The conditions of these varieties, in part understood [229], are in part unintelligible; and hence vocal resonance should never be trusted to alone in the diagnosis of a cavity. The form of resonance most strongly distinctive of an excavation, is *whispering* pectoriloquy: but cavities may exist without this; while resonance of the sort may exist in dilated bronchi surrounded by hardened tissue [667].

961. A cavity of large size, with hard and smoothish walls, and containing thin fluid in moderate quantity, may emit a guggling sound when the trunk is abruptly shaken. The heart's action, too, if the cavity be near, produces a similar sound of cardiac rhythm.

962. Where great loss of substance is produced in the right lung by excavation, and its remaining tissue is much reduced in bulk, the heart may be drawn greatly out of its place, and beat to the right of the sternum.

963. Before we examine the physical signs of *arrested* and *retrogressive* tuberculisation, a subject almost completely new, it will be advisable, the essential anatomical conditions, presumed to accompany such suspense or retrogression, be clearly understood: with this view they may be set down as follows:—

Arrested Tuberculisation.	*Retrogressive Tuberculisation.*
First Stage.—Amount of deposit originally small; ceases to take place; tubercle remains as such; collapse of cells and lobules; obstructed bronchial tubes on minute scale; local atrophy of parenchyma; local emphysema.	*First Stage.*—Absorption of tubercle, previously calcified, or sometimes, in all probability, independently of any such change; [*] much bronchial obstruction; local collapse and atrophy; local emphysema.

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[*] Cyclop. of Anat., art. Products, adventitious, p. 108.

Arrested Tuberculisation.	*Retrogressive Tuberculisation.*
Second Stage.—Reduced bulk of affected part by contraction of induration-matter, hardening, puckering of surface and substance; collapse and atrophy of parenchyma; emphysema.	*Second Stage.*—Induration, contraction, collapse, atrophy of parenchyma; union of broken surfaces by exudation-matter, producing linear or irregularly puckered cicatrices, provided the breakages of lung-substance have been on a very small scale.
Third Stage.—Cavities of various size continue to secrete pus or muco-pus; bronchial tubes closed beyond them; collapse of communicating tissue; the cavities are lined with a pseudo-mucous membrane.	*Third Stage.*—Great reduction of bulk of excavated portion of lung, substance of which, where not excavated, is more or less replaced by induration-matter; more or less deep indentations of pulmonary surface by contracting fibro-plastic-matter in pleura; more or less marked reduction of area of cavities, but doubtful if actual closure occurs, where the original excavation has been on a large scale.

964. Local suspension or retrogression of tuberculous disease is, at one or other stage, and for a shorter or longer period, one of the most common occurrences in its evolution. I hold, indeed, that evidences of such retrograde action may invariably be found in the lungs of persons cut off by chronic phthisis. But this is a very different thing from admitting that pulmonary consumption thus frequently stops in its onward course. For, the truth is, that on the one hand, while action of curative import takes place in one part of a lung, fresh deposition of tubercule or fresh breaking up of tissue, may be advancing in another; and, on the other hand, the duration of the stationary or retrogressive state is often so short, that clinically speaking, the tuberculised patient is scarcely a gainer.

965. The physical signs of arrested and retrogressive tuberculisation vary so widely, not only with the stage and conditions of the disease actually present at the moment suspension of its progress was effected, but also with the length of time that has intervened between such suspension and examination of the

chest, that it is impossible to establish precise general rules on the subject. I shall consequently simply put down a few specimens of the conditions that have fallen under my notice in particular cases.

966. Notable depression, supra- and infra-clavicular and supra-scapular, imperfect expansion, very weak, harsh respiration, dulness under percussion, exaggerated fremitus, and strong vocal resonance. Here softening signs on a small scale had existed eighteen months before.

967. Similar depression, imperfect movement and dulness, dry clicks, with deep inspiration, which is weak and harsh. Here softening signs had existed only six months before.

968. Dulness under percussion at the apex, feeble bronchial breathing, with a deep-seated creaking sound in inspiration [217]. Here, nine months before, softening signs were actively present, and both the local and general symptoms seemed to promise a rapidly fatal issue. I have not seen this patient professionally since the above observation was made, and cannot say what the local state may now be (seven months later); but I know by report that her general health is excellent, and that there are no chest symptoms, except occasionally slight cough.

In these three instances suspension, and more or less perfect retrogression, in the second stage, seems to have been accomplished. Here is another example. A girl aged 15, one of whose sisters had died of rapid consumption under my care at Brompton, presented in July 1850, scrofulous keratitis, enormous cervical glands, the signs of enlarged bronchial glands, with percussion-dulness at the left apex, front and back, bronchial respiration, excess of vocal resonance and large-sized thin liquid rhonchus, purulent expectoration and notable loss of flesh.* In 1853 this patient was kindly sent to me by Dr. Routh, who had accidentally seen her, with all the attributes of health, her glands in a perfectly natural state, and, with the exception of slightly weak respiration at the left apex, not a single morbid physical sign.

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* A. Petrolini, U. C. H., Females, vol. v. p. 175.

969. Dulness under percussion below the clavicle, with slightly tubular quality, hollow dry cavernous respiration, vocal resonance diffusely exaggerated: I have recently seen a case with these signs, where, from the account given me, I infer that four months ago the progress of phthisis was most active. At a later period, the signs may be much the same. Thus I saw in May last, a girl (E. Blow) whom I had first known with excavated lung, six years before, and whose history pointed to seizure yet four years earlier, in whom the signs of excavation still existed in the form of hollow respiration with tubular percussion-note; but there was no rhonchus, and the chest had lost somewhat of the flattened phthisical look habitual to it for years.

970. Respiration of laboured and jerking rhythm, weak and bronchial, but without clicks or rhonchal sound of any kind, and with very slightly deficient resonance under percussion. Here there had been, two months earlier, much greater dulness under percussion, respiration of diffused blowing type and cogged-wheel rhythm,—and the general symptoms of advancing tuberculisation. Congestion had probably disappeared, and the tuberculous deposit become quiescent.

971. Lastly, I have known some instances where the physical signs of induration had existed at either apex to a slight amount, in individuals belonging to a tainted family, where the local and general symptoms of phthisis had made their appearance, and where the signs in question totally disappeared along with the symptoms, leaving behind them merely trivial harshness in the respiratory sounds. These I believe to have been examples, not only of tuberculisation, but of phthisis, retrograding in the first stage: a very striking probable illustration of such course will by and by be dwelt upon.

972. *Symptoms.*—The symptoms of phthisis do not admit of such systematic division into stages as its physical signs: there

is no symptom absolutely peculiar to any one of the three stages. It will be better, therefore, to consider them *seriatim*, under the heads of *local*, *general*, and *incidental*.

973. *Local Symptoms.*—Cough, unless in latent cases, is an essential symptom; habitually unpreceded by coryza, rarely paroxysmal, it is at first either dry (but very probably in not more than about one-tenth of cases) or attended with colourless, frothy, watery, or mucilaginous-looking expectoration, the last somewhat characteristic. The thinner fluid at an early period sometimes deposits a thicker, grumous-looking substance in small quantity, resembling the deposit in barley-water: I do not believe that this occurs except in phthisis. Gradually the expectoration grows glairy and mucous, striæ of opaque buff or pale yellow appearing on a lighter ground; it grows less and less aërated: presently the sputa become purulent, either in the form of small pellets with jagged, sharply cut outlines, opaque, semi-floating, non-aërated, and of yellowish colour (boiled-rice sputum), or of larger masses with ragged edges, or of broad, flat, discoid lumps, darkish green in tint, quite smooth in outline, and remaining apart if expectorated into water. Eventually the sputa acquire an ash colour, run together in one mass, totally free from air, and are constituted essentially of pure pus: this condition of sputa is of very bad augury, if it occur in a patient under treatment; when found in persons who have greatly neglected themselves up to the time of seeking advice, it is generally rapidly modified by medical care. Occasionally sudden profuse expectoration of purulent fluid occurs, in some cases traceable to abrupt evacuation of a cavity, in others to abundant secretion from the walls of old cavities and neighbouring bronchi. The quantity of expectoration varies greatly in a mass of cases: sometimes profuse, especially in the earlier and closing periods, it may be very slight throughout; and I have known cases run their course without any expectoration at all. I once saw a lad, aged fifteen, admitted into hospital with typhoid fever, become tuberculous during

convalescence, and, dying within a short period, present large cavities in his lungs, though he had never, as far as could be learned by constant questioning, expectorated a single sputum: he must, of course, have swallowed them all, as *children* almost invariably do.

974. Hæmoptysis, including under this term even expectoration simply streaked with blood, is a symptom of extreme frequency, occurring, as I formerly found, (Brit. and For. Med. Chir. Rev., January, 1849) in about 81 per 100 of cases.

The following general inferences are derived from the examination of 106 cases:—

"Hæmoptysis was of very slightly (4 per 100) more frequent occurrence in males than females.—Hæmoptysis to a medium amount is about four times less common than to very slight or to profuse amounts,—both taken together. Very profuse hæmorrhage from the lungs is more common in males than females.—Medium frequency of recurrence of hæmoptysis is materially less common, and this in both sexes, than a single or than repeated attacks. Further, repetition of hæmoptysis is more common in males than in females.—It is materially more common for a first hæmorrhage to be more profuse than subsequent ones, than for subsequent ones to be more profuse than the first.—Hæmoptysis is more frequently met with (and this independently of any influence of duration of the disease) in persons who have reached the second and third stages, than in those whose lungs have not yet softened. This proposition is more markedly true of males than of females.—There does not appear to be any notably greater tendency to hæmoptysis, where the right lung has reached a more advanced stage than the left, nor *vice versâ*, where the left has taken the lead. It seems improbable that either lung is more effective in causing hæmoptysis than its fellow.—The frequency of hæmoptysis increases with advancing years in both sexes. The increase is more abrupt in females than in males, and in the former appears connected with the catamenial function.—This greater frequency of hæmoptysis in persons of more advanced years, does not depend altogether on greater duration of the disease; for those who had had hæmoptysis, had been phthisical for only a mean period of eight months longer than those who had not spit blood.—The most common periods for the occurrence of hæmoptysis, were, at the very outset, or after the expiration of the first month; it is very rare for hæmoptysis to occur within the first month, unless it has actually appeared as the first, or among the first symptoms. The phrase "first symptom" here is to be understood with a qualification to be by and by explained.—In upwards of half the cases of notable hæmorrhage (beyond 4 oz.) this occurs, or has occurred, as the "first symptom," corroborating the inference as to the excess of amount of first over subsequent hæmorrhages.—Hæmorrhage of this amount is rare as a coexistence with other first symptoms (in $\frac{1}{13}$ of these cases) appreciable by the patient.—Streaked or tinged sputa are, on the contrary, of very common appearance amongst the earliest symptoms.—But streaked or tinged sputa are rarely, or never, *the* "first symptom" singly and alone.—Season does not appear to exercise any marked influence on the occurrence of a first hæmorrhagic attack.—Hæmoptysis never appeared as the *bonâ fide* first symptom in these cases, the phrase being understood in its absolute sense without qualification; it is so only in the sense that it is the first symptom particularly noticed by patients—the first occurrence that leads them to watch their health."

975. The last proposition is important: it leads one to reject, on clinical grounds, the old notion, that phthisis is really caused by hæmoptysis in those cases where it *appears* to lead the way in the train of morbid events,—in other words, to deny the existence of "phthisis ab hæmoptoe."

976. The quantity brought up, at any one time, varies between a few streaks and some pints of pure unmixed blood.

977. The prognosis of tuberculous hæmoptysis is a subject of great interest. Hæmoptysis may kill *directly* or *indirectly*; but my analysed series of 131 cases of phthisis furnishes but two examples of such mode of death. In one, death was direct, but from asphyxia, not loss of blood: the trachea and bronchi, as far as traceable, were plugged with coagula. In the other, death occurred from exhaustion five days after the hæmorrhage. In these and some other fatal cases that have fallen within my observation in private practice, the patients have invariably been *males*; in this point of view, as well as in certain others, hæmoptysis is a more serious event in men than in women. A first hæmorrhage having been severe, it is unlikely that a subsequent one will kill *directly*; for it is not often that a recurring hæmoptysis is more severe than a first severe one, granting that they do not follow so closely upon each other as to appear a mere continuation of one and the same attack. One of the cases above referred to constituted an exception to this rule. It appears from the cases I have

examined, that hæmoptysis is frequent in proportion to the duration of the primary disease; if so, the converse of the proposition cannot be evaded; and we are forced to conclude that *frequently-recurring hæmoptysis does not reduce the mean duration of life, after seizure with tuberculous symptoms, in any given mass of cases.* It is not repugnant to reason to admit, then, that in a certain number of instances hæmorrhage from the lungs may act as a local therapeutical agent. No doubt the disease sometimes runs on more rapidly after an attack of hæmoptysis; but I cannot help regarding the two things as mere coincidences.

978. The tints in phthisical hæmoptysis vary,—venous red is rare; florid red most common: sometimes the sputum is of dark brick-red hue; again, of a light flesh-like pink.* Moulded clots, even of minute size, are excessively rare [575].

979. The odour of phthisical expectoration is nauseous, *sui generis*, not actually fœtid. It may accidentally acquire the wet-plaster like, or putrid smell of gangrene, under circumstances already described [862].

980. In the course of phthisis calcareous particles and masses, merely gritty or of petrous hardness, and in size from a pin's point to a pea, may be expectorated. I have known this continue for years steadily or interruptedly. But while such expectoration is a clear proof of local retrogression, the disease may in other parts of the lung be actively advancing.

981. The microscopical elements of phthisical sputa are very numerous. First, epithelium tesselated, cylindrical and ciliated from the bronchial tubes; salivary fluid, and epithelium from the mouth. Secondly, blood-disks, (even when no reddish tint exists to the naked eye,) melanic cells and molecules, molecular fat, oil globules, and saline matter, crystalline and amorphous. Thirdly, exudation-matter in patches, exudation-cells, and pus-cells. Fourthly, fragments of pulmonary fibre, capillary vessel and nerve. Fifthly, dark molecular matter, soluble neither in

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* Tagg, U. C. H., Females, vol. ix., p. 108.

ether nor in hydrochloric acid, and probably tuberculous,—and in very rare cases, cells apparently those of tubercle: I have, at least occasionally, seen in the opaque buff-coloured striæ of comparatively clear sputa, cells non-nucleated and more angular in outline than those of exudation-matter. Sixthly, the vibrio lineola and mycodermatous entophytes.

Now the presence of fragments of tissue indicates breakage of the lung-substance, and may furnish its earliest evidence. The existence of tubercle-cells, if certain, is of course distinctive of phthisical disease. Otherwise the characters enumerated have no precise diagnostic signification.

982. Of the chemical characters of the sputa I know little. Popular prejudice, looking upon the change from saline to sweet taste as of evil import, is probably well founded; for in the outset the sputa, essentially those of catarrhal flux, are markedly saline; with the advance of the disease and obstruction of lung they may become saccharine. Sugar, however, is by no means always to be found.*

983. The cough in phthisis more frequently brings on vomiting or nausea, than in any other pulmonary affection: if a fit of coughing occurs shortly after a meal, more or less of this is habitually vomited in the majority of cases that have reached the second stage.

984. Dyspnœa is the reverse of a prominent symptom. The frequency of respiration rarely reaches any notable height in the pure disease; and when the act is morbidly accelerated, the pulse also beats with such increased frequency, that the ratio of the two suffers scarcely any change. I have seen few cases

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* Thus the sputa of Nettleship, U. C. H., Males, vol. x., p. 146, and of M. Harris, U. C. H., Females, vol. ix., p. 326, both in the third stage, were boiled for twenty minutes with two parts of distilled water, then treated with acetate of lead and soda; the filtered fluid gave no evidence of sugar with Trommer's, Poggiale's, or the bichloride of tin tests. These results, carefully obtained by my clinical clerk, Mr. W. T. Coster, were confirmed by actual analysis in the Birkbeck Laboratory of University College.

of very marked dyspnœa, of which the patients volunteered complaints. In these cases there was either great emphysema, with bluish lividity of the face, and general coolness of surface, or nothing could be detected except the tuberculous disease to account for the symptom.

985. Actual pain, or distressing sensations within the thorax, exists in the great majority of cases; but severe suffering of this kind is the exception. The pain may in part be intra-pulmonary, true pleurodynic, intercostal-neuralgic, or pleuritic. Local pleurisy is by far its most frequent tangible cause; to local evanescent dry pleurisies are in the main due the wandering chest-pains of these patients, and to more permanent pleuritic changes their comparatively fixed sufferings. But, as already shown [673], the mechanism of pleuritic pain itself requires elucidation.

986. *General Symptoms.*—Febrile action, sometimes attending the very earliest local symptoms (in sixteen of ninety patients observed by Louis, excluding cases of acute phthisis), does not appear, in the majority of instances, till softening has occurred. Actual shivering, or merely chilly sensations, with subsequent heat, followed by perspiration, constitutes a complete paroxysm. The shivering is slight, as a rule,—the perspiration profuse. The chest and head are in many cases the chief seats of perspiration. Night-perspirations, especially towards morning, are common, even where there is no distinct febrile paroxysm. Sudamina are rare. There may be two daily paroxysms,—a morning and an evening one.

987. The pulse, habitually, but not invariably, frequent, is of wide range,—within my own observation, from 60 to 140 per minute in uncomplicated cases. The frequency of the pulse sometimes changes remarkably in the same case within a day or two: occasionally an obvious cause, in the shape of some inter-current attack, can be found for this,—quite as commonly it baffles explanation. It has been said the increase of frequency on changing from the recumbent, to the sitting and standing, postures is less

than in other diseases attended with debility: the general statement seems not unfounded, but exceptions are frequent.

988. The digestive organs suffer more or less in the majority of cases. Thirst is rarely absent through the whole course of the disease, whether the alimentary canal be free from anatomical change or not. Pain and tenderness below the ensiform cartilage, with loss of appetite, nausea, and occasional vomiting, indicate sub-inflammatory action in the mucous membrane of the stomach, and can scarcely be considered direct symptoms of the primary disease. Vomiting of food, brought on by fits of coughing, and unaccompanied by any other gastric symptom, does not indicate any textural change in the organ.

Stomatitis, simple or diphtheritic, is not uncommon towards the close of the disease: epiphytic formation occurs, but rarely, in the exudation. Chronic pharyngeal abscess is in some instances met with,—in one case of the kind, that fell under my notice, produced by caries of a cervical vertebra. I once saw uncontrollable frothy salivation form a prominent and distressing symptom of the closing period, without obvious affection of the buccal membrane or salivary glands.

In 1847, M. Fredericq drew attention to a red streak at the edge of the gum opposite the lower, and sometimes also the upper, incisors, as one of the earliest signs of phthisis,—the colour in highly febrile phthisis being brick-red, in hæmorrhagic phthisis blue. Subsequently he professed, that in the latter period of all chronic diseases a blue or red streak will be found. M. Vanoye, in turn, describes a white marginal line; * and Dr. Thompson insists upon the red. I believe with M. Fredericq, that the red line is to be found in a variety of chronic blood-diseases, but more frequently in phthisis than others. It may be completely absent, however, to the last hour; and is notably more frequent in patients of the lower than of the upper ranks, —a fact to be explained in some measure by habits of cleanliness and the reverse.

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* Ranking's Retrospect, vol. xii., p. 218.

The state of the bowels varies greatly,—in some rare cases they continue natural, in a certain number constipated even to the last hour, in others relaxed from time to time, in a fourth class permanently loose. Diarrhœa may depend merely on secretive changes in the bowels, on small ulcerations in the ileum and jejunum, or on extensive destruction of the mucous membrane of the colon, as well as of the ileum. In the first case, the symptom is temporary, and easily controlled; in the second, more persistent, and with difficulty arrested: in the third case, absolutely unmanageable. In the last condition, as insisted on by M. Louis, the motions are very numerous, reddish, or putty-coloured, fluid, and of putrid odour. If the rectum be ulcerated, the motions may be pseudo-dysenteric.

It is important to remember that ulcerations may exist in the ileum without pain or tenderness: again, I have known, in a case running an acute course, marked abdominal pain and tenderness conjoined with obstinate constipation, where after death, in spite too of the frequent use of purgatives, the bowels contained abundant solid fæces, and the ileum was extensively tuberculised and ulcerated.*

The glands of Brunner are sometimes enlarged and tuberculised: sub-acute duodenitis follows, and doubtless plays a part, though an uncertain part, in the phenomena of phthisical dyspepsia.

989. The external lymphatic system, on the whole, is rarely tuberculised in the adult. I have known the cervical and axillary glands, greatly enlarged in phthisical people, rapidly fall to the natural size without suppuration or symptom of any kind. Severe lumbar neuralgia may be produced by infiltrated glands pressing on the nerves: but, common from cancer, this is rare from tubercle. Disease of the mesenteric glands, tuberculised in from one-third to one-fifth of phthisical adults, takes part

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* Hodson, U. C. H., Males, vol. ix., p. 16. Even the large, as well as the small, intestines may be ulcerated, and yet no diarrhœa exist. Plimpton's Case, Lancet, loc. cit., p. 579.

in perverting nutrition,—but in what form and to what extent remains to be discovered. The bronchial glands, often moderately enlarged by tubercle, itself quiescent or retrograde, rarely induce symptoms in the adult [635—649].

990. Slight wandering at night, on waking out of sleep, sometimes occurs; with this exception the mental faculties retain their clearness in the majority of cases to the last few hours—trifling failure of memory, and inability to follow a train of thought, alone existing towards the close. Not only this, but the perceptive and reasoning powers sometimes acquire unwonted vigour and acuteness, as the disease advances; and the imagination and fancy grow unnaturally vivid,—the individual becomes, as it were, idealised.

The temper, though irritable, is singularly hopeful. Everyone has seen cases in which arrangements for future years are made within a few days of death; and I have actually known the question of a change of profession complacently considered within *three hours* of the fatal event. There may be, in such cases, an effort on the part of patients to deceive themselves and those about them, as to the real state of things: but, nevertheless, hopefulness constitutes a special clinical feature of the disease, and cannot by any means always be explained by the absence of pain. How strong the contrast between the phthisical and the cancerous patient in this point of view! When marked cerebral symptoms exist, they always indicate intercurrent disease.

991. The cellular tissue remains remarkably free, as a rule, from serous infiltration. If there be marked œdema even of the ankles, there is almost invariably some morbid state, besides phthisis, to account for the fact; *à fortiori*, if there be general anasarca of one or both lower extremities. The latter state, of great rarity, depends on coagulation of the venous blood, either spontaneous or produced by phlebitis.

992. Pains in the lower extremities are sometimes, towards the close of life, the prominent symptom: if co-existent with

anasarca, the state of the veins explains them; under other circumstances they seem simply neuralgic.

993. Emaciation ranks among the most constant and most striking phenomena of phthisis. The alterations in the adipose tissue are the same as in other forms of emaciation,—the cells diminish in size, oil gives place to watery fluid, and crystalline separation of the fat-elements takes place within them.

Preceding, as emaciation does, in some cases all other symptoms, local or general, the presence of tubercles in the lungs (or the constitutional state, of which those tubercles are the local evidence), obviously acts as an efficient cause of wasting,—loss of appetite, vomiting, diarrhœa, and perspirations, are subsidiary in their influence. It is not only the external fat and cellular tissue that waste: the muscles, and the parenchymata suffer too; as proved by Louis and Bizot, the weight of the heart and calibre of the aorta are less in the victims of phthisis than of any other disease, except cancer. The weight of the body, as a whole, consequently diminishes, rapidly, surely, and progressively, more especially as no serous accumulations form to give fictitious weight. But there is a curious fact connected with the emaciation of phthisis, and, for aught I know to the contrary, of other chronic diseases: namely, that it is not an invariably steady process. I have repeatedly found, by placing patients in the balance within short intervals, that there are rises and falls in weight, intercurrent to the general progressive tendency downwards, and occurring irrespectively of any obvious changes in diet, appetite, or colliquative symptoms. The late Dr. Robert Williams, of St. Thomas's, as I several years ago learned from his friend, Dr. Silvester, inferred from a large mass of observations upon this point, that there was a law of periodicity regulating the rises and falls of weight. I have no means of ascertaining what the period established by Dr. Williams was; and my own observations are too limited to supply the deficiency. I feel tolerably sure that the interval is shorter than a month—the period at which Sanctorius, generalising from experiments on his own person, inferred that a passing increase of two pounds' weight (ascribed to lunar influence!) took place in health.

Irritability of the muscles under the influence of percussion or other physical stimulus, is carried perhaps to a higher point in the emaciation of phthisis than of other chronic diseases.

994. But in some instances phthisis runs its course almost to the end without notable emaciation: the body may be plumply fat, while large cavities exist at the apices, and the disease is advancing downwards. Such retention of fat generally indicates a sound state of the alimentary canal; still I have known plumpness maintained with feeble appetite, and occasional diarrhœa.* On the other hand rapid emaciation may take place in persons eating abundantly, and free from a trace even of dyspepsia.

The thoracic coverings waste most, the facial fat least, rapidly; exceptions to this rule are very rare.

995. Bulbousness of the finger-ends; curvation and transverse cracking of the finger-nails, and falling of the hair, are observed in a certain share of cases.

996. M. Louis has arrived at the conclusion, contrary to what had previously been held, that the sexual appetite in the male undergoes impairment. Menstruation, he found, ceased in the female, when the total duration of the disease was under a year, at about the middle of its course; when the duration of the disease ranged between one and three years, the catamenia continued commonly to appear till the last third. Menstruation may remain natural to the last; or disturbances in point both of time, quantity, and quality, may occur. The uterus and ovaries present no deviation from structural health, as a necessary condition of phthisical paramenia. Neither is it possible to explain, by the course or predominant symptoms of the disease, the healthy or perverted state of the function.

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* J. Gonner, Females, Consumption Hospital, Chelsea. This patient retained flesh, until Bright's disease, which rapidly destroyed her, supervened.

997. It has been generally supposed that pregnancy retards the progress of phthisis,—the disease acquiring increased activity after parturition. I have positively observed several cases in which many of the symptoms of phthisis became less prominent during pregnancy. This might be the fact, it is true, without the real pulmonary disease being suspended in progress: but it is curious that I do not remember to have opened, or to have seen opened, the body of a female dying of phthisis, and at the same time pregnant. My sphere of observation is, however, not the most favourable for encountering pregnant women. M. Grisolle has lately examined these questions, and comes to the conclusion that the disease is somewhat increased in rapidity of progress during pregnancy; while after parturition, it is slightly mitigated, or at least, remains stationary. The number of cases he has collected (and all other persons that I know of are in the same predicament) are quite insufficient to solve the problem.

998. I some time since numerically examined the question, whether the tuberculous diathesis intensifies or weakens the force of fecundity in the female, and of the procreative faculty in the male, and was led, by the facts, to the conclusion, *that the procreative power of phthisical males is below the average—the fecundity of phthisical females materially above it.* Taking the two sexes together, and regarding them as phthisical stock prepared to propagate, the female activity is counterbalanced to a certain extent by the male inactivity; 11·82 years of phthisical cohabitation produced a mean of 0·83 children less than 17·48 years of non-phthisical cohabitation.*

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* For the facts themselves, *vide* Medical Times, July 6, 1850. The above results accord with M. Louis's general statement, concerning the failure of sexual inclination in the male; they are totally at variance with an assumption of M. Grisolle, that conception is rare in phthisical women. The total number of cases on which my inferences are founded, is 91 of phthisical, 220 of non-phthisical, persons.

999. The blood in the early period of phthisis is deficient somewhat in red corpuscles, and very slightly in fibrine; the proportions of albumen and water are increased; the serum appears to be less alkaline than in health. With the advance of the disease the blood becomes hyperinotic, presumably from the irritating influence of the tubercles and intercurrent inflammations. Quite at the close the fibrine and the solids generally undergo notable diminution. There is no microscopical character, that I know of, in the red corpuscles, which can be trusted to as the slightest guide in the diagnosis of the disease.

1000. Dr. Hope says, he has seen many cases of phthisis in which coagulation of the blood occurred, from mere sluggishness of motion, in the femoral vein, with œdema of one or both extremities. I have met with but one example of death from phthisis, with coagulation of the blood, and obstruction of the veins in a lower extremity; in this instance, the femoral and iliac veins of the right limb were *inflamed* in the most positive manner.* I cannot help doubting that Dr. Hope's observations can be frequently verified; still I have within the last two years twice observed œdema of the legs in the course of phthisis, disappearing rather rapidly under treatment, and unattended with any great tenderness in the track of the femoral veins. The œdema occurred here independently, in all probability, of phlebitis; yet there was positive venous obstruction in the limbs,—whether produced by sluggishness of current, and hyperinotic constitution of the blood, I am unable to affirm.†

1001. In the early stage, when the local symptoms are inactive, and marked fever absent, the urine presents no seriously abnormal characters. When the pulmonary texture breaks up, and fever runs high, the urine becomes small in quantity, of strong odour, deep urinous colour, and of high, or rather high, specific gravity [1028, 1022]. If the system be impoverished by great loss of blood, or colliquative drain of any kind, the secretion becomes pale, watery, and of low gravity. As a rule, in the active disease, the uric acid ranges more or less above the average of health. Temporary increase of urea may occur, but even where waste of tissue is rapidly going on, and the patient takes nitrogenised food in good quantity, the daily average may fall to about forty or fifty grains,—Lehmann's average for healthy male adults being 255 grains. Oil globules are very rarely to be found in phthisical urine; I have never detected them, except where the patient was taking cod-liver oil. Albumen sometimes appears passingly in minute quantities, either probably from indigestion, or renal congestion. In a certain proportion of cases, all the symptoms of Bright's disease supervene. Simple pyelitis, with acid urine, deposit of pus, and of casts of the tubuli, with such amount only of albumen as is referrible to the liquor puris present, occurs in rare instances; in yet rarer the evidences of tuberculous pyelitis, as proved after death, may be obtained during life.[*] The urine occasionally contains minute quantities of sugar[†] a fact interesting in connection with the well-known tendency of glycohæmia (saccharine diabetes) to terminate in phthisis. On first thought, the existence of so readily oxidisable a product as sugar in the urine, would seem subversive of Liebig's hyperoxidation theory of phthisis; but the fact that in saccharine diabetes, gum and alcohol are completely oxidised, while sugar is not, shows that an elective power of oxidising some, and not all, oxidisable materials, exists in the economy. Sugar may be, and commonly is, totally absent from the urine in the most advanced cases of phthisis.

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* Henry James, U. C. H., Males, vol. v., p. 130, 1850.

† The influence of heteræmia is well shown by Mackenzie.—Med. Chir. Trans., vol. xxxvi.

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[*] Wright, U. C. H., Females, vol. viii., p. 195. "Deposit in urine: pus-corpuscles, oil globules, amorphous granular organic matter, cells larger than blood disks, smaller than pus-cells, with granular contents, somewhat irregular and angular outline, and showing no nucleus under acetic acid." One of the pyramids, infiltrated with tubercle, had ulcerated into the renal pelvis, which contained 1 oz. of pus and tubercle-detritus.

[†] Arthur, U. C. H., Females, vol. vi., p. 51, Feb. 1851. First stage of phthisis passing into second; specific gr. of urine=1024; weight of patient= 8 st. 3 lbs. on Feb. 19; =8 st. 8¾ lbs. on March 20, when the sugar had disappeared, the patient having meanwhile taken oleum morrhuæ in small doses.

Micturition is of natural frequency; but female patients are often tormented by slight involuntary discharge of urine during fits of coughing.

1002. *Incidental Symptoms.*—The incidental symptoms of phthisis are the clinical expressions of its "secondary morbid changes." A case of phthisis may run its course without a single one of these ever occurring, or they may occur in various combinations and variable intensity. Sometimes these symptoms are so severe as to throw into the shade those of the pulmonary disease, and monopolise the attention both of patient and physician: while the secondary changes causing them, sometimes seem to accelerate, sometimes retard, the advance of the primary affection, and prove themselves the real causes of death.

1003. Pneumonia frequently occurs in the course of tuberculous disease, or at its close. In the former case, it is either a mere local effect of the progress of tuberculisation, or it may be extensive, and acquire almost the importance of an idiopathic attack of the disease. But even then it is rarely of serious augury: it is, singularly enough, less fatal than primary pneumonia. What I have observed on this head, is in perfect accordance with the observations of M. Louis;—the mean duration of the inflammation, even, is less than when occurring in sound lungs. Some of the most marked examples of rapid resolution I have met with, were in phthisical persons. [795.]

M. Louis holds that pneumonia occurring at the closing periods of phthisis, is almost of necessity fatal. But, admitting this, it does not follow that there is any special tendency in the phthisical to death by pneumonia. In point of fact, pneumonia—or conditions of the lung referred to that disease—is a tolerably common appearance in the lungs of persons cut off with all varieties of chronic maladies. The proportion of cases in phthisis is scarcely greater; and where so-called hepatisation is found, it has not always been the actual cause of death.

Pneumonia limited to the *anterior* portion of either apex, is, in the great majority of cases, tuberculous,—not invariably so, however. I have known pneumonic signs limited to the infra-clavicular region, without being the result of tuberculous irritation, so far as the disappearance of all vestige of morbid signs at the apex can be accepted as proof of this.

1004. Pleurisy we have already met with in the dry and exudation forms, as an attendant on tubercle. Effusion occurs in a fair proportion of cases, and is always a most serious complication: complete recovery is singularly rare. Double pleuritic effusion, occasionally met with, is, as shown by M. Louis, in some measure peculiar to phthisis. Effusion, single or double, not unfrequently hastens the fatal issue.

1005. Bronchitis, local or general, invariably occurs in the course of phthisis. The form most peculiar to the disease seems to be that limited to one apex or to one base.

1006. Ulcerations of the epiglottis give rise to great dysphagia, especially of liquids, which frequently return by the nostrils. There is fixed pain opposite the affected part.

1007. Chronic inflammatory changes in the larynx are indicated by change in quality of the voice and cough, which grow hoarse, muffled, and cracked; and in proportion as ulceration destroys the chordæ vocales, the voice degenerates into a hoarse whisper. I have never observed absolute aphonia. Pain, stinging, pricking, or shooting, is more or less constant; and a distressing sensation of dryness is experienced. If the epiglottis be free, there is no dysphagia. The physical signs are rough, coarse respiration in the larynx, with sonorous, sibilant, or thin gurgling rhonchus, according to the dryness or moisture of the diseased surfaces.

1008. I know of no positive symptoms of ulceration of the bronchi; those of chronic tracheitis are obscure. Pain, heat, and dryness, with choking sensation above the sternal notch, are all that I have observed; and these symptoms may exist without tracheal ulcers, while ulcers may exist without them. Intense, so-called tracheal dyspnœa, I have not met with.

1009. The symptoms of perforation of the pleura are elsewhere described [741]. The perforating process may make its way, through both adherent pleuræ, without pneumothorax, to, or through, the skin,—and be followed, or not, by subcutaneous emphysema.

1010. Of abdominal incidental symptoms, those indicating chronic peritonitis are the most important. Enlargement of the abdomen, pain and tenderness under pressure, ascites, and tympanitic distension of the intestines, are the prominent symptoms. The ascites may rapidly disappear under treatment, while the tympanitis remains, and the outline of the intestines appears on the abdominal wall. Pain may be constant, or occur only at the moments flatus moves in the bowels. All control over escape of flatus may be lost; probably, by a consensual arrangement for the avoidance of pain, the effort, necessary for its retention, is omitted. Diarrhœa is present in the majority of cases, whether the bowel be ulcerated or not. This secondary affection may fall into a quiescent state; but if so, the pulmonary disease unfailingly, or almost unfailingly, grows more active.

1011. Fatty disease of the liver, not a common secondary change in this country, has no special symptom that I know of. Probably it affects the properties of the fæces. Its physical signs are those of simple enlargement of the organ.

1012. Fistula in ano is, according to my observation, more frequently met with, in males especially, than it has of late been the habit to believe.

1013. Serious as are the evils of ulcerations of the bowels, those of cicatrisation may be more so: death may, in fact, be the result of accompanying contraction of the bowel. M. Louis reports a case, where the effects of intestinal stricture, the earliest indications of disturbed health, observed by the patient, proved fatal in about twenty-two months, having throughout kept the chest-symptoms in abeyance, though cavities existed in the lungs.

1014. The existence of tubercle in the meninges is only known through inflammation of the textures in which it is seated. The symptoms of tuberculous meningitis in the adult are, in the main, as follows. Cephalalgia, most commonly frontal, and vomiting, flushing and pallor of the face, followed by delirium, commonly of the quiet kind, very rarely boisterous or violent, paralysis, hemiplegic or partial, twitchings of the face, strabismus, somnolence, and eventually coma: the pupils, at first commonly contracted, eventually dilate; the pulse and respiration both slacken; the skin becomes cool; the cough and other chest-symptoms disappear. Remission of the cerebral symptoms, simulating convalescence (though I have never seen it so marked in the adult as in the child), may occur towards the fatal issue, which generally takes place in from seven to eighteen days. Whether recovery is possible, will be considered hereafter. I have now observed at least six cases of this affection in the adult, in which a peculiar form of mutism formed a striking symptom. The patients, when questioned, looked steadily in the speaker's face for a few moments, and then, without making the slightest effort at speech, deliberately, but without any sign of petulance, turned their heads away.

1015. Deafness, depending on tuberculous destruction of the membrana tympani, is noticed in some cases.

1016. *Course.*—Chronic phthisis may, especially in females, for a portion or for the entire of its course, whether this be of medium or considerable duration, remain symptomatically *latent.* That is, tubercles may exist in the lungs and slowly work out their ill influences on the organism, through secondary blood-changes, without awakening attention by any of their ordinary local thoracic symptoms, such as cough, expectoration, pain in the chest, and dyspnœal sensations.

1017. Four classes of cases may be met with referrible to this head. To the first belong instances in which violent hæmoptysis, or perforation of the pleura, are apparently the first symptoms of the disease,—the former commonly so, the latter very rarely so. To the second belong cases of slow course in which one of the secondary morbid states, such as chronic peritonitis, or ulcerative diarrhœa, mask or really suspend the progress of primary mischief. In a third class we find cases, where an individual is generally out of health, without suffering from local or general symptoms of any severity. In a fourth rank cases where very prominent symptoms exist, such as emaciation, fever, loss of appetite and sleep, relaxation of the bowels without apparent cause,—none of them of obvious pectoral origin.

1018. The local latency of tuberculous disease in some of these cases seems explicable on the principle of *Duobus* morbis *simul obortis, vehementior obscurat alterum.* But in instances where none of the secondary morbid states exist, the fact baffles explanation. The great points for the observer to bear in mind are, that, while such latency is not only a real, but a frequent, clinical fact, physical signs alone can disclose the true state of things. A single tap above the clavicle will sometimes give the ready clue to what has hitherto been utterly mysterious. Let him not be diverted from his belief by the assurances of patients that they have never coughed,—the assurance will occasionally be given by persons who, at the moment they give it, have *cavities in their lungs.*

1019. *Diagnosis.*—Although the diagnosis of phthisis can be made, with surety, through the physical signs and local symptoms alone, there are some other conditions, which may have more or less subsidiary force in doubtful cases. One of these is the existence of hereditary or family taint. But with what frequency may we expect aid in diagnosis from the existence of such taint? Singularly enough it is impossible to reply fully to this query at the present day. Some years since I made an attempt to determine the frequency of hereditary transmission of phthisis, by comparing two series of persons, the one phthisical, the other non-phthisical, and the final conclusion to which the analysis of 446 cases led was, "that *phthisis in the adult hospital-population of this country is, to a slight amount only, a disease demonstrably derived from parents.* There is no reason to believe that the law differs among the middle and higher classes of society; but to warrant an absolute assertion, we should require an analysis of two series of cases (similar to those I have now adduced) which had been collected among those classes." (B. & F. Med. Rev. Jan. 1844). But my cases throw no light on the amount of transmission in children,—in whom hereditary influence is probably more widely traceable.

1020. Can the relative progress of tuberculisation in the two lungs be employed as an element of diagnosis? The following results of the analysis of 143 cases of phthisis throw some light on this question:

> "From these propositions it follows that the disease attains the softening point with not very unequal rapidity in the two lungs. Such difference as exists signifies that the point is more quickly attained in the left than in the right organ; for it appears that softening of the left lung, the right being still firm, was more common (under two distinct points of view) in the ratios of 29·51 to 27·12, and 24·13 to 19 51. I have, however, no means of ascertaining satisfactorily whether this depends on deposition being on an average earlier in the left lung than in the right, while the actual rapidity of subsequent destruction is equal in both; or from the destructive process being more rapidly effected on the left side, deposition being coeval on both sides.
>
> But, as will appear from the subjoined comparison, the laws regulating the disease in males and females probably differ in this respect: they certainly did in these 143 cases.

Tabular comparison of the condition of the two lungs in (143 *cases*) *of both sexes.*

MALES.	FEMALES.
(*a.*) *Right lung:* 24 in first stage, 4 in which the *left* had reached the	(*a.*) *Right lung:* 37 in first stage, 14 in which the *left* had reached the

second or third stage,—that is in 16·67 per cent. of the number.

(*b.*) *Right lung:* 56 in second or third stage, 11 in which the *left* still remained in first stage.—that is, in 19·64 per cent. of the number.

(*c.*) *Left lung:* 31 in first stage, 11 in which the *right* had reached the second or third,—that is, in 35·49 per cent. of the number.

(*d.*) *Left lung:* 49 in second or third stage, 4 in which the *right* still remained in the first,—that is, in 8·16 per cent. of the number.

second or third stage,—that is, in 37·83 per cent. of the number.

(*b.*) *Right lung:* 26 in second or third stage, 5 in which the *left* still remained in first stage,—that is, in 19·27 per cent. of the number.

(*c.*) *Left lung:* 28 in first stage, 5 in which the *right* had reached the second or third,—that is, in 17·86 per cent. of the number.

(*d.*) *Left lung:* 35 in second or third stage, 14 in which the *right* still remained in the first,—that is, in 40 per cent. of the number.

The propositions marked (*b*) do not tell in one direction or the other; those marked (*a, c, d*) depose strongly to the greater rapidity of evolution of the disease in the right lung of males, and in the left of females. Let it be remembered, too, that in the only two instances in which either lung was wholly unaffected, it was the *left* lung of *males* that had escaped. Why the disease should advance more quickly on the right side in men, and on the left in women, does not clearly appear: if it be suggested that the greater frequency of pneumonia of the right lung in the male will explain the fact of its becoming a more rapid prey to tuberculisation, the difficulty is to explain why the left should be the earlier victim in females."

1021. I will now embody in a series of propositions some of the most interesting combinations of diagnostic conditions, of which I have actually seen more or less numerous examples.

(*a*) A young adult, who has had an obstinate cough, which commenced without coryza and without any very obvious cause, a cough at first dry and subsequently attended for a time with watery or mucilaginous looking expectoration, and who has wandering pains about the chest, and loses flesh even slightly, is in all probability phthisical. (*b*) Should there have been hæmoptysis to the amount of a drachm even, the diagnosis becomes, if the patient be a male and positively free from aortic aneurism, mitral disease or hypertropy of the right ventricle, almost positive. (*c*) If, in addition, there be slight dulness under percussion at one apex, with jerking or divided and harsh respiration, while the resonance at the sternal notch is natural, the diagnosis of the first stage of phthisis becomes next to absolutely certain. (*d*) But not absolutely certain: for I have known every one of the conditions in *a*, *b*, and *c* exist (except hæmoptysis, the deficiency of which was purely accidental), where one apex was infiltrated with encephaloid cancer, and no cancer had been discovered elsewhere to suggest to the physician its presence in the lung. (*e*) If there be cough, such as described, and permanent weakness and hoarseness of the voice, the chances are very strong (provided he be non-syphilitic) that the patient is phthisical. (*f*) If decidedly harsh respiration exist at the left apex or at the right apex behind, if the rhythm of the act be such as I have called *cogged-wheel*, and there be dulness, so slight even as to require the dynamic test for its discovery, there can be little doubt of the existence of phthisis. (*g*) If with the same combination of circumstances deep inspiration evokes a few clicks of dry crackling rhonchus, the diagnosis of phthisis, so far as I have observed, is next to absolutely certain. (*h*) If these clicks on subsequent examination grow more liquid, the transition from the first to the second stage may, as a rule, be very surely announced. But all the conditions of *f*, *g*, and *h*, may be simulated in infinitely rare cases of combined local simple consolidation and bronchitis: the adjoining induration gives bronchial rhonchi a clear ringing quality. (*i*) If there be slight flattening under one clavicle, with deficiency of expansion-movement, harsh respiration and slight dulness under percussion, without the local or general symptoms of phthisis, the first stage of tuberculisation cannot be diagnosticated with any surety, unless there be incipient signs at the other apex also: the conditions in question limited to one side might depend on chronic pneumonia or on thick induration-matter in the pleura. (*k*) The existence of limited, though marked, dulness under one clavicle, with bronchial respiration and pectoriloquy, so powerful as to be painful to the ear, the other apex giving natural results, will not justify the diagnosis of phthisis. I have known this combination of signs, where the apex of the lung was of model health, but a fibrous mass, the size of a walnut, lay between the two laminæ of the pleura. I would even go further, and say that the combination in question is rather hostile than otherwise to the admission of phthisis; as, had tuberculous excavation formed at one side, the other lung would, in infinite probability, have been affected with the disease in an earlier stage. (*l*) Pneumonia, limited to the supra- and infra-clavicular region on one side, and not extending backwards, is commonly, but not always tuberculous. (*m*) Subcrepitant rhonchus, limited to one base posteriorly, is not, as has been said, peculiar to tubercle; it may exist in emphysema and in mitral disease. (*n*) Chronic peritonitis, in a person aged more than fifteen years, provided abdominal cancer can be excluded, involves as a necessity the existence of tubercles in the lungs. To this law of M. Louis, it is necessary to add the qualification, provided Bright's disease (and this is readily ascertained) be also absent. But the question of cancer is far from easily settled: thus there may be pain and tenderness, evidently peritonitic, ascites varying in amount from time to time, now a relaxed, now a somewhat obstinately constipated state of bowels, vomiting more or less persistent, extreme emaciation, diphtheritic stomatitis, percussion-dulness in the supra-spinous fossæ, muco-purulent expectoration, no cancerous growth discoverable, and all this in a young female, and yet the peritonitis shall be cancerous, the lungs and bronchial glands being the seat of calcified tubercle only. The tint of skin may be somewhat cancerous, the face emaciated as much as the rest of the body, the peritonitis may be limited, the ascites trifling, and night-sweats and hæmoptysis absent, it is true, in such a case; but to form a positive diagnosis on these points would be rash.* (*o*) Pleurisy with effusion, which runs a chronic course in spite of ordinary treatment, is in the majority of cases, tuberculous or cancerous: the character of the symptoms, previously to the pleurisy, will generally decide between the two. (*p*) Double pleurisy, with effusion, is not, as has been said, significant of tubercle; for it may depend on Bright's disease. If the latter affection can be excluded, carcinoma and pyohæmia remain as other possible causes. (*q*) If a young adult, free from dysentery, and who has not resided in tropical climates, suffers from obstinate diarrhœa, which goes on month after month, with slight remissions or intermissions, even though there be no cough, he is in most strong probability phthisical. If physical signs, to the slightest amount, exist at either apex, he is, almost to absolute certainty, phthisical. (*r*) If a young adult, free from secondary syphilis and spermatorrhæa, and not dissolute in his habits, steadily lose weight, without clear cause, he is in all probability phthisical, even though no subjective chest-symptoms exist. (*s*) But he is not by any means certainly so; for he may have latent cancer in some unimportant organ, or he may have chronic pneumonia. (*t*) Nay, more, he may steadily lose weight, have dry cough, occasional diarrhœa, and night-sweats, and present dulness under percussion, and bronchial respiration under both clavicles, and yet be non-phthisical. I have known all this occur in cases, both when the lungs were infiltrated superiorly with primary encephaloid cancer, and when they contained secondary nodules of the same kind. (*u*) Failure of weight becomes less valuable as a sign of phthisis, the longer the thirtieth year has been passed. (*v*) Percussion-dulness at both supra-spinous fossæ, cough, abundant muco-purulent expectoration, hæmoptysis (frequently free from bistre-colour tint, though sometimes presenting this [882]), great emaciation, inexplicable except on the hypothesis of tubercle, curved nails and bulbous finger-ends, may all of them exist, and yet not a gray granulation even exist in the lungs. Heart disease, chronic pneumonia of the base and pulmonary apoplexy may alone be present.* (*w*) The discovery of cardiac disease with marked symptoms deposes against, but does not exclude, the existence of active tuberculisation. (*x*) The existence of cancer in any organ is unfavourable to the presence of tuberculous disease; but tubercule and cancer *may* co-exist, even in the same lung. (*y*) Tubercle very rarely, active phthisis still more rarely, if ever, co-exists with leucohæmia. (*z*) Constant vomiting of food with the cough, the stomach being sound, though much more common in phthisis, may attend chronic bronchitis. (*a a*) Light hair and eyes furnish no argument in favour of phthisis in a doubtful case; the unpublished investigations of Dr. John Beddoe, undertaken on logical principles, tend rather to show that persons with dark hair are more liable to phthisis, than those who are fair or xanthous. (*b b*) If a person seen for the first time and examined with the spirometer, blow a number of cubic inches exceeding the alleged average of men of his height, the inference, that therefore his lungs are sound, is unjustifiable; they *may* at the very moment be excavated by tubercle. If, conversely, an individual fail to blow up to, or even nearly to, his physiological standard, his lungs may nevertheless be perfectly sound. And granting that the failure shows unsoundness of lungs, it throws no light on the nature of the pulmonary disease. They who maintain that phthisis can thus be diagnosticated, may, it is true, refer to examples of successful diagnosis on this principle; but, simply on the doctrine of chances, he must often prove correct, who, given a chronic pulmonary affection, pronounces it at once to be tuberculous,—such is the vast preponderance of that form of disease.

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* All these facts were illustrated by the case of Scott, U. C. H., Females, æ'atis 29, vol. ix., pp. 5—101; the left lung weighed only 9, the right 12 oz. Almost precisely the same conditions existed in a young lady, aged 18, (seen with Dr. Neil Arnott and Sir J. Clark,) destroyed by abdominal encephaloid.

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* Lewis, U. C. H., Females, vol. ix., p. 342.

1022. Patients frequently attach importance to the determination of the apex most affected. This point may be settled on different principles by different persons. The amount of consolidation in a given spot will guide one, the superficial area of consolidation another, the uniformity of consolidation or the stage of the disease, others. Hence arise sometimes apparent differences of opinion between observers, where none really exist. The disturbing influence of emphysema, and the fact that the disease may suddenly become most active at the apex hitherto most quiet, must not be forgotten.

1023. I would here offer, for the sake of beginners, a few cautions in the application of physical diagnosis in phthisis. Never attempt to give a positive opinion as to the actual state of the lungs, where there has been recent hæmoptysis, or when pleuritic effusion, bronchitis, or pneumonia is present: I of course refer to cases where there may, or may not be, signs of the first stage; if excavation exist, its signs may be generally unravelled in spite of these complications. Always examine the supra-spinous fossæ, as well as the clavicular regions. Trust very little, if at all, to the conditions of vocal resonance; accept with great caution the evidence of slight changes in respiration, unless they be corroborated by percussion-changes; place no confidence in jerking respiration (even though local) in a hysterical woman,—nor in harsh respiration, with its prolonged expiration, limited to the right apex in any woman, nor in very very slight dulness at the right front apex in man or woman. Lastly, never give a confident opinion, in a nicely-balanced case, from a single examination; make examinations in various postures; and always compare carefully with physical signs the local and constitutional symptoms.

1024. *Mode of Death.*—There is a gradual and normal death from phthisis by asthænia,—itself induced by exhausting discharges, waste of fluids, insomnia, constant wearing suffering of various kinds, and, often in spite of free consumption of food, imperfect reparation. Or such gradual extinction may be brought to a more or less rapid close by ulceration of the bowels and profuse diarrhœa; obstruction of the intestine by cicatrisation of ulcers; chronic peritonitis; double pleurisy with effusion; perforation of the pleura; pain, irritation and dysphagia from epiglottic and laryngeal disease; hæmoptysis indirectly by anæmia; Bright's disease; and tuberculous meningitis. Or death may take place suddenly and unexpectedly; and then the occurrence may, as M. Louis has well distinguished, be explicable or inexplicable. In the former category appear deaths by hæmoptysis from actual loss of blood or asphyxiating obstruction of the air-tubes; extensive sudden pneumonia of adynamic character; or œdema of the glottis. In the second category the fatal issue is hypothetically referred to "fatigue of the heart," or to general reduction of the consistence of the brain.

1025. *Duration.*—Of the duration of a disease, which destroys life at all periods, between twenty days and twenty years, little of practical applicability can be said. Of 307 cases observed by M. Louis, the following arrangement may be made:—

4	died within	1 month.	98	died within	6 months.
15	„	2 months.	160	„	9 „
26	„	3 „	264	„	24 „

Hence, at the end of two years, 43 of 307 patients only survive; so that at the time of seizure in any given case the chances are about as 6:1 that death will ensue before the commencement of the third year; the chances are only as 2:1 in favour of passing the sixth month; while those of surviving the ninth are against the sufferers in the ratio of 160:147, or closely as 1·09:1. These estimates refer to the period of first declaration of symptoms. They refer, also, to past experience: there is every reason to believe that the introduction of cod-liver oil into practice has improved the chances of phthisical life.

1026. Season does not exercise the kind of influence which might be anticipated in shortening the career of the consumptive sufferer. It may be calculated from the Registrar General's Twelfth Report that the mortality in London during the five years 1845-1849, averaged in the quarter ending March, 1774, in that ending June, 1762, in that ending September, 1593, in that ending December, 1573. So that the mean deaths in the six coldest months rate at 3347, in the six warmest 3355. Compare these results with those concerning pneumonia [192]!

1027. *Treatment.*—Experience shows that the treatment of the phthisical may, with legitimate confidence, aim at either maintaining a *status in quo,*—at producing slight local and general improvement, or marked improvement of this kind,—at effecting a total removal of all subjective symptoms, while the physical signs remain partially active,—or at accomplishing total removal of the symptoms, and bringing about a quiescent state of the physical signs, while the general health, weight, and vigour, have improved to such an extent, that the patient shall believe himself totally free from disease, and that the medical observer might be disposed to share his opinion, did not passive physical changes remain. I say medical art may legitimately aim at these ends, because, on the one hand, these ends have been actually obtained; and, on the other, the man has not yet appeared who can point to results more perfect than the best of these, as the positive, direct, and ordinary effect of any known system of medication. This latter clause is not in the least at variance with the well-known fact, that phthisis sometimes spontaneously undergoes permanent suspension of its course.

1028. The following results, which I obtained at the Consumption Hospital, justify the foregoing statements, and furnish guides to *prognosis*:—

1. Of a given mass of patients entering the hospital in all stages of the disease, and in every variety of general condition — between the actually moribund state and that of but slight constitutional suffering—the number leaving it, on the one hand, *improved* or *unadvanced* was more than double that, on the other hand, leaving it in *a worse state* or *dying within its walls* (the exact ratio is 67·84 : 32·16). If the cases in which death was actually imminent at the period of admission, were excluded, the result would be very materially more favourable than this. 2. In 4·26 per cent. of the cases, complete restoration to health, not only as regards apparent disturbance of the functions generally, but as regards local evidence of active pulmonary disease, was effected. 3. Complete removal of symptoms was more frequently effected in the male than in the female; but, on the other hand, the results were, on the whole, slightly more favourable in the latter than in the former sex. 4. All patients whose condition grew worse, while they were in the hospital, had reached the stage of excavation on admission; and all patients, whose

tubercles were yet unsoftened on admission, left the hospital either improved, or having had a *statu quo* condition kept up. Improvement is more probable than the reverse, even where excavation exists on admission. 5. In a given mass of cases, the chances of favourable influence from sojourn in the hospital will be greater, in a certain, undetermined, ratio, as the duration of the disease previous to admission has been greater,—in other terms, natural tendency to a slow course is a more important element of success in the treatment of the disease, than the fact of that treatment having been undertaken at an early period. 6. The mean length of stay in the hospital in the most favourable class of cases, nearly doubled that in the least favourable. 7. The chances of benefit are more in favour of those whose trades are wholly or partially pursued out of doors. 8. The results did not appear to be influenced by the laborious or non-laborious character of the trade individuals might have pursued. 9. The age of the sufferers did not exercise any very material influence on the character of the results. 10. Patients coming from the country have, on an average, a slightly stronger chance of improvement, than the residents of London and the suburbs. 11. Patients admitted during the warmer half of the year, benefit by a sojourn at Brompton, to a slight extent, more than those received during the six colder months.

1029. My task in examining the efficacy of various *specific* agents will be brief. Iodide of iron, chloride of sodium, liquor potassæ, chlorine and iodine inhalations, hydrocyanic acid, creasote, digitalis, are disposed of in the masterly analyses of their claims by M. Louis; and naphtha may be allowed to remain in the rather rough grasp of the British and Foreign Medical Review. But cod-liver oil cannot be so lightly dismissed.

1030. I began to employ the oil at the Consumption and University College Hospitals nine years ago, urged to the step by the strong advocacy of Dr. Hughes Bennett, and took an early opportunity of testifying to its remarkable powers in tuberculous and other scrofulous diseases.* The conclusions at which I have arrived concerning its use in phthisis, are as follows :—

1. That it more rapidly and effectually induces improvement in the general and local symptoms than any other known agent. 2. That its power of

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*. Nature and Treatment of Cancer, p. 202, 1846.

curing the disease is undetermined;—I mean here, by "curing" the disease, its power of causing, along with suspension of progress, such change in the organism generally, as shall render the lungs less prone to subsequent outbreak of tubercles, than after suspension occurring under other agencies.* 3. That the mean amount of permanency of the good effects of the oil is undetermined. 4. That it relatively produces more marked effects in the third, than in the previous stages. Opinions the most diverse have been held on this point; M. Taufflied † taught that it had little or no effect on phthisis, if at all advanced; M. Péreyra ‡ *reduced the size of cavities in a few weeks* by its administration. 5. That it increases weight in favourable cases with singular speed, and out of all proportion with the actual quantity taken;—that hence it must in some unknown way save waste, and render food more readily assimilable. 6. That it sometimes fails to increase weight. 7. That in the great majority of cases, where it fails to increase weight, it does little good in other ways. 8. That it does not relieve dyspnœa out of proportion with other symptoms. 9. That the effects traceable to the oil in the most favourable cases are: increase of weight, suspension of colliquative sweats, improved appetite, diminished cough and expectoration, cessation of sickness with cough, and gradual disappearance of active physical signs. 10. That in some cases it cannot be taken, either because it disagrees with the stomach, impairing the appetite (without itself obviously nourishing), and causing nausea, or because it produces diarrhœa. 11. That in the former case it may be made palatable by association with a mineral acid; and in the latter prevented from affecting the bowels by combination with astringents. 12. That intra-thoracic inflammations and hæmoptysis are contra-indications to its use, but only temporarily so. I have repeatedly given the oil within a day or two of the cessation of hæmoptysis without any return taking place. 13. Diarrhœa, if depending on chronic peritonitis, or secretive change, or small ulcerations in the ileum, is no contra-indication to the use of the oil; even the profuse diarrhœa caused by extensive ulceration of the large bowel is not made worse by it. 14. That the beneficial operation of the oil diminishes, *cæteris paribus*, directly as the age of those using it increases,—a singular fact, which probably may one day, when the textural peculiarities of youth and age are better understood, aid in giving a clue to its mode of action. 15. That the effects of the oil are more strikingly beneficial, when a small extent of lung is implicated to an advanced stage, than where a relatively large area is diseased in an incipient stage. 16. That when chronic pleurisy or chronic pneumonia exists on a large scale, the oil often fails to relieve the pectoral symptoms. 17. That it often disagrees, when the liver is enlarged, and probably fatty. 18. That weight may be increased by it, and yet the local disease go on. 19. That weight may increase, the cough and expectoration diminish, night-sweating cease, the strength which had been failing remain stationary, under the use of the oil, and yet the local disease be all the while advancing. I have known softenings on a small scale pass in two months into tolerably extensive excavation under these circumstances,—singular proof of the nutritive powers of the agent.

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* That such cures really occur in rare instances (and they are as perfect as in any other organic disease, when they do occur) is indubitable. It has been the vanity of late years to deny this absolutely, because a scientific (or pseudo-scientific?) explanation of the fact cannot be found. I am not one of those who refuse to accept the evidences of my senses, because I am unable to comprehend what they teach me, and in this matter echo the sentiments of the physician in a recent French tale, speaking of a phthisical recovery:— . . . . "ces miracles de guérison, aux quels la Science ne croit pas, faute de les comprendre, *et devant lesquels je me prosterne, en priant la bonne et sage Nature d'en être moins avare.*"—Pierre, par Madame Reybaud.

† Gaz. Méd. de Paris, Nov., 1839.

‡ Du Traitement de la Phthisie. Bordeaux, 1843.

1031. Demonstration will probably be by and by obtainable, from the returns of the Registrar General, of some diminution in the annual mortality from phthisis. Without claiming for the oil the power of curing the disease, we still must look for this result; for indubitably it does possess the power of prolonging phthisical life,—an effect which must of course lessen the annual number destroyed by the disease.

1032. Of the three kinds of oil,—the brown, light brown, and pale—the brown, I believe, as matter of actual experience, to be the most efficacious.* But, though taken greedily by infants, it is more distasteful than the pale to the adult palate, and hence in grown persons I have been forced to use the latter, less active kind (in fact *gild the pill*), in order to ensure oil being swallowed at all. Chemists give no positive answer to the question, on what depends the efficacy of the drug? Its influence on the composition of the blood is yet undetermined. A single analysis by Simon shows a state of hypinosis, combined with a great excess of albumen, may follow on its use; the solid constituents generally were in large amount. The patient had been bled repeatedly for hæmoptysis. The iodine and bromine of the oil, its phosphorus, butyric acid, gaduine, biliary material, and its mere fatty matter have been severally accorded the chief part in the beneficial results. Recently the presence of phosphorus has been denied by M. Personne; and M. Winckler seems to maintain that the efficacy of the oil depends on its having propyl for its radical, instead of glycyl, the ordinary basis of oils. The iatro-chemical discussions on the subject do little more than exhibit the existing poverty of our knowledge of the intimate action of remedies. On the other hand, the established efficacy of the oil—a substance of which *à priori* views would scarcely have admitted the possible retention by the phthisical stomach—is another of the conquests of *experimental* therapeutics.*

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* On the properties of these varieties of oil, see De Jongh on Cod-liver Oil by Carey, Lond., 1849.

1033. The dose of the oil at the outset should never exceed, often fall short of, a drachm twice daily: it may be taken in water, milk, orange wine, infusion of quassia or chiretta, weak brandy and water, or any aromatic water agreeable to the patient. The dose may be gradually raised to half an ounce, twice, or at most thrice, in the twenty-four hours. I have never seen any good, and often observed ill, effects follow the attempt to pour in large quantities. If there be disposition to regurgitation of the oil, the whole daily quantity may be taken with advantage at bed-time.

If the stomach reject the oil absolutely, it may be administered in enemata, combined with an astringent, or by inunction. I have, however, found that the rectum grows irritated too soon to allow of sufficient continuance of the enemata; through the skin, the oil acts more efficaciously.†

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* As is well known, cod-liver oil has been a household remedy from time immemorial in the north of Europe. To the extensive use of fish-oil may probably be due the singular immunity from phthisis enjoyed by the Icelanders in spite of the anti-hygienic conditions in which they live. See Schleisner, B. and F. Med. Chir. Rev. April, 1850.

† The Romans ranked the process of oiling themselves, and then basking

1034. The attempts to prove almond oil and neats-foot oil as efficacious as the oleum morrhuæ, seem to me to have failed. Skate-liver oil is probably largely mixed with the commercial cod-oil,—and is by some held to be medicinally quite as active. Some patients, to whom oil is unbearable, digest cream well: a pint to a pint and a half has been taken daily without gastric disturbance, and with manifest increase of flesh in advanced cases of the disease under my direction.

1035. On iatro-chemical grounds phosphate of lime has been recommended of late: satisfactory clinical evidence in its favour has yet to be furnished; nor does the theory seem very profound which assumes that, because retrogressive tubercle calcifies spontaneously, ergo, if calcareous salts be taken into the stomach in active phthisis, advancing tubercle will assimilate them, and forthwith retrograde.

1036. The *hygienic* management of consumptive patients is of extreme importance. Plain nutritious food, regularity of hours of meals, attention to the state of the digestive organs, moderate and frequent exercise in the open air, either active (riding and walking), or passive (carriage, swinging, or yachting), the cold, tepid or warm bath, friction of the surface, the use of flannel next the skin, loose lacing of the stays in females, exercise of the respiratory muscles and lungs by deep inspirations, reading aloud, movements of the arms, gentle gymnastic exercises, are all of essential service in the early period of the disease, and especially while the diathesis, rather than the local mischief, claims attention. Exchange of profession or trade, from the sedentary and laborious to those moderately active physically, and but slightly taxing the intellect, is advisable.

1037. As matter of experience, change of climate unquestionably proves, in many cases, when resorted to in the early

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in the sun, among their most important hygienic luxuries. The reader will remember the reference to the habit in the letter of Pliny the Younger to Tacitus, describing the catastrophe of Pompeii and the death of his uncle, the Elder Pliny.

period of the disease, of fundamental service. Transference to a more genial atmosphere than that to which the invalid has been accustomed, in some rare instances permanently arrests the disease; in others prolongs life; and in a third class relieves symptoms strikingly, and renders the close of existence comparatively comfortable. True, there is a fourth series of persons, whose sufferings are aggravated, and end hastened, by the toil of a journey, and the want of home luxuries and associations. But these are cases in which disorganisation is at once extensive and active in the lungs, while the general symptoms indicate that profound constitutional disturbance, which no art can rectify. In the selection of a climate for any particular case, the dry or moist character of the attending bronchitis, the natural liking of the individual for relaxing or bracing air, and the general tendency to the *strictum* or the *laxum* in the organism, furnish the best guides. (See Appendix.)

1038. The winter may be passed without danger in-doors in England in an artificial climate, provided proper precautions be taken in regard of ventilation. Air heated to a fixed temperature,* and perpetually renewed by a scientific system of ventilation, such as Dr. Arnott's, forms an excellent substitute for the naturally soft air of milder climates than our own. If, on the one hand, there be the drawback of want of open-air exercise, there are the counterbalancing advantages of the comforts and familiar friends of home, and the escape from the labour of travelling. Nor can I avoid referring to the prospect of advantageous winter exercise in a genial, yet not over-close, atmosphere, held out to the consumptive invalid by the permanent erection of the Crystal Palace at Sydenham.

1039. The *palliative* treatment of phthisis is a subject so vast, that I can here merely enumerate some of the chief agents that have appeared useful in this regard. Counter-irritation, in the various forms of repeated flying blisters, ammoniated liniments, croton oil, the strong acetic acid and turpentine, are important remedies: tartarised antimony is the least valuable agent of the class. Special irritability of skin and very great emaciation are the only barriers to the employment of such counter-irritation, which I prefer as a rule to permanent blisters, issues or setons under the clavicle. The addition of tincture of iodine in variable proportions has appeared to me beneficial; and a blistering alcoholic solution of iodine and iodide of potassium, cautiously applied, appears not only to relieve pain, but actually to promote absorption,—at least I have found the respiration and percussion-sound improve locally under its use.

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* Some latitude may be allowed, of course, for individual taste; but, as I found from questioning upwards of one hundred patients at the Brompton Hospital, an atmosphere at all below 64° Fah. is disagreeable to the majority.

1040. Although sceptical as to the solvent theory of its action, I have seen benefit derived from liquor potassæ combined with a sedative and bitter tonic. Digitalis and hydrocyanic acid are both useful in cases where the heart is irritable,—checking sometimes, indeed, to a very remarkable degree, various symptoms dependent on that irritability. Where a general state of erethism exists, opium, in some of its forms, must be employed, independently of necessity for it as a hypnotic or anodyne. I have never observed results justifying faith in iodide of potassium as an internal remedy; but the syrup of the iodide of iron, especially if there be the least anæmia, is a valuable medicine; it does not increase fever, and sometimes enables a patient to bear cod-liver oil, who had previously failed in the attempt to take this. Mineral acids, with light vegetable bitters, improve the appetite, and control undue action from the skin. Quinine is rarely borne well, and produces no specific effect on the hectic fever.

1041. Whatever the theory of Broussais may plead to the contrary, general experience recognises not only the inutility, but the actual mischief, of bleeding, general or local, with the idea of curing consumption. True, intercurrent congestions and inflammations in the thorax may require, both for themselves and to avert their possible consequences, slight abstractions of blood; but it is remarkable how effectually minor attacks of the kind may be counteracted by dry-cupping, blisters, and small doses of antimony.

1042. The treatment of phthisis by daily emetics (supported by a peculiar, but not altogether sound, theory of the site occupied by tubercle) cannot, I fear, appeal to rigid experience in its favour,—though strongly recommended by Giovanni de Vitis, Carswell and Todd Thomson. Unquestionably it does less mischief, however, than the morbid anatomy of the stomach in this disease would lead us to expect.

1043. I have no experience of dry inhalations, whether of oxygen, hydrogen, or carbonic acid; and but little of moist inhalations, chloruretted or ioduretted: the latter, I have recently found, relieve some forms of phthisical bronchitis. The inhaled vapour of warm water, impregnated with emollient herbs, such as *althæa officinalis*, narcotic extracts, or creasote palliates cough, dryness of throat, and laryngeal irritation.

1044. The secondary conditions of phthisis often require special treatment. *Hæmoptysis* has already been spoken of. The relief of *cough*, which has resisted opiates and ordinary medicines, may often be effected by the application of three or four leeches above the sternal notch.* *Dyspnœa* may be mitigated by inhalation of small doses of chloroform.† Naphtha sometimes succeeds, sometimes fails, in diminishing *expectoration:* when successful, hæmoptysis may be the immediate result. *Nausea and vomiting* may be controlled by effervescing draughts, Seltzer water, plain iced water, by prussic acid, by a combination, which I have found very useful, of creasote, stramonium and hop; if there be acidity, by liquor calcis or liquor potassæ; by blisters and sinapisms; or, if there be tenderness, by leeches to the epigastrium. *Diarrhœa* may be arrested by soothing laxatives (rhubarb with magnesia, or castor oil, cajuput, and tincture of opium) when dependant on irritant matter in the bowel. Sometimes diarrhœa, without evidence of inflammatory action, is more or less amenable to the whole class of astringents,—among which I may specially refer to sulphate of copper and opium, gallic acid and acetate of lead; and enemata of starch and laudanum, or of hæmatoxylon, krameria, or tormentilla. If there be evidences of sub-inflammatory action, friction of the abdomen with rubefacients, or the application of a few leeches either to the tender part of the abdomen or to the anus, sinapisms, blisters, and emollient poultices, are advisable. The hip-bath is very hazardous. Where the large bowel is extensively ulcerated, remedy after remedy will probably be tried in vain. I have seen some benefit even in these cases from the sulphate of zinc in two grain, and the nitrate of silver in one grain, doses: enemata of the latter, in solution, are also sometimes useful. In all forms of diarrhœa, except when it has become confessedly irremediable, the diet should be low and as dry as possible: attendant thirst may be mitigated by dissolving small pieces of Wenham-lake ice in the mouth. *Perspiration* may be controlled by avoiding much drink and using light bed-clothes; the chest may be rapidly sponged at bed-time with tepid vinegar-and-water, or with decoction of oak-bark. Bark and the mineral acids, gallic acid and lead, have, as medicines, the most perceptible effect. *Inflammatory and ulcerative changes in the larynx* may be stayed by local leeching, blistering, counter-irritation with equal parts of spirits of turpentine and croton oil,* or an issue to the side of the neck or nucha. Some benefit, even temporary improvement in the voice, occasionally follows stimulation of the internal surface of the larynx, with strong solution of nitrate of silver (one or two drachms to the ounce of water); but I have never seen more than temporary benefit from the process in phthisical laryngitis. *Chronic peritonitis* requires the application of leeches, and blisters, which may be dressed with morphia, when the pain is severe, and friction with iodurctted liniments; emollient poultices with laudanum, if not too thick and heavy, relieve pain. If there be much ascites, diuretics are indicated; but they often fail. In point of fact, this is one of the conditions of tuberculisation the least controllable by art; though it sometimes undergoes a questionable kind of spontaneous cure. Cod-liver oil, if not already in use, should, of course, be administered: the existence of diarrhœa is not a contra-indication. *Tubercular meningitis* is yet more difficult of cure; moderate local bleeding, cold irrigation of the head, purgation, and other revulsive measures, and calomel internally, are alone to be trusted to. I have as yet seen but one even apparent example of recovery in an adult, where the symptoms of tubercular meningitis seemed established: a few prominent particulars of the case may here be given.

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* Hall, U. C. H., Females, vol. ix., p. 128.

† S. Wells, Med. Times, Oct. 11, 1851.

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* This combination must be very cautiously used,—three or four drops only being rubbed in with a piece of flannel for a single minute at a time.

John Stoner, ætat. 25, admitted U.C.H. for second time, in third stage of phthisis, April 10, 1849. The chest-disease gradually advancing.—*June* 2. Loss of power of speaking, semi-stupor, with difficulty roused, dysphagia, drink pouring back through mouth, but not nose, pupils sluggish, no distinct convulsion, lower extremities drawn up, no paralysis, sensibility blunted, fixed frontal cephalalgia; seems conscious of all that is passing around him; P. 102, R. 44.—*June* 4. Since this seizure cough almost gone, scarcely any expectoration; alternate pallor and flushing of cheeks; no rigidity or convulsions, no screams, no strabismus; complete insensibility of dorsum of hands; tendency to rigidity of knee-joints; fingers firmly clenched, when unclenched by bystander contract again; P. 142, R. 44.—*June* 8. Sensibility returned, still speechless.—*June* 9. Spoke as usual for first time. This man was treated with mercurials, but not to ptyalism. Discharged on July 13th, he was readmitted August 18th, and died of his pectoral disease, the cerebral functions being perfectly natural, on October 11th, 1849. *Post mortem:* No morbid appearance at fissures of Sylvius; over both anterior lobes fine florid injection, numerous granules, opaque, greyish-white, size from pin's point to pin's head, lie under cerebral arachnoid of these lobes; convolutions here appear drawn together, as if from deep-seated contraction in the sulci; membranes here generally opalescent; dura-matral arachnoid adherent posteriorly to cerebral arachnoid of left hemisphere; membranes thicker than natural; anterior convolutions at the curve forwards, especially at left side, somewhat opaque; those of left side not materially softened,—those of right firmer than natural.

I have since found calcareous matter in the pia mater of the sulci in a person cut off by phthisis.*

My actual experience on the question whether *fistula in ano* should be cured in phthisical patients, is small; such as it is, it deposes emphatically against interference, unless (what is very rare) there be wasting discharge and serious suffering.

1045. The diet of phthisical patients should be nutritious and non-stimulant,—wine and spirits being, in ordinary cases, avoided. I have, however, in special cases of *non-febrile* phthisis, with cold skin, lividity of face, and dyspnœa, prescribed brandy medicinally, with the good effects of raising the temperature and relieving the dyspnœa. Perhaps the notion that brandy supplies pure aliment for respiration may explain the fact, that habitual spirit-drinkers (*placed otherwise under favourable hygienic influences*) do sometimes suffer less, and live longer, with excavated lungs, than sober persons. I have seen some three or four most remarkable examples of the kind,—examples, indeed, which first led me to a cautious use of brandy medicinally. Gum Arabic, another aliment of respiration, is not open to the objections that attach to alcoholic fluids, and should be allowed largely as an article of food. The mucilaginous material of the Carragheen and Iceland mosses is useful, as satisfying appetite somewhat without exciting the pulse. As respects the quantity of animal food allowable, no general rule can be laid down; so much as each stomach can digest, without local suffering or systemic disturbance, may be safely permitted. Without being an advocate of the "mutton-chop and porter" plan, I am deeply convinced that a low diet is seriously injurious. Light bitter ale, if it do not excite cough, may be permitted in small quantity at dinner. Sarsaparilla agrees with many stomachs, as a diet drink.

1046. The habit of smoking, if indulged in with moderation, has not appeared to me specially injurious in phthisis: it is not impossible, indeed, that the infinitely minute quantity of nicotin inhaled, may exercise a locally soothing effect. But the profuse use of tobacco is decidedly injurious to the health of the phthisical, as of the non-phthisical.

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* Osmond, U. C. H., Males, vol. iv., p. 355.

ACUTE PULMONARY CONSUMPTION.

1047. When phthisis proves fatal in from twenty days to ten or twelve weeks from the first appearance of symptoms, it may fairly be said to have run an acute course.

1048. I have seen, in cases of death under these circumstances, the three following anatomical states; (*a*) limitation of tuberculous deposit, softening and excavation to the apices, just as in ordinary chronic phthisis,—no peculiarity existing in the anatomy of the disease to account for the rapidity of death,—which, however, I have not observed, in this form, earlier than the eleventh week; (*b*) general accumulation of crude tubercles, or tuberculous infiltration of lobules, through both lungs, with irregular softening, small excavations existing in various parts, and patches of hepatisation presenting themselves here and there (*acute softening form*); (*c*) general studding of both lungs with semi-transparent grey granulations, coupled with the first stage of pneumonia, that of bright arterial injection, or with hepatisation (*acute miliary form*). The forms *b* and *c* may coexist in the same lung.*

1049. Now, in the first case (*a*), there is nothing peculiar in the signs or symptoms; the progress of the disease, except in regard of its rapidity, is exactly the counterpart of that observed in chronic phthisis; and a lurking suspicion frequently remains in the observer's mind, that tubercles may have existed in the *latent* state, for a greater or less time, before the outbreak of symptoms. In the second and third cases, there is not only a still greater rapidity of course (death may occur in less than three weeks from the outset), but the signs and symptoms are peculiar, and far from being as significant of the existing disease as might be wished.

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* Knowles, U. C. H., Males, vol x., p. 18.

1050. *Physical signs.*—In the case (*b*) where the lungs are more or less crammed with softening tubercle, the physical signs are as follow :—Inspection discloses nothing special; the semicircular width of the mainly affected side, especially if pneumonic infiltration has occurred, may be slightly increased; application of the hand may detect some increase in vocal vibration,* and some deficiency of chest motion ; but it is impossible to say in what part of the chest this deficiency may be most marked. The resonance under percussion is more or less impaired,—at first in some limited points, subsequently over the surface pretty generally; but to the last hour various spots may give resonance not positively abnormal. The quality may be markedly tubular in some places, independently of excavation : with cavity, even on a small scale, amphoric. The inspiration weak in some points, exaggerated in others, assumes bronchial or even faintly tubular quality over the most densely consolidated parts ; but the highly marked sniffling metallic breathing of hepatised consolidation may be wanting, even where the entire lung is very closely infiltrated with softening tubercle,†—doubtless because the air is never so completely expressed from the vesicles in the latter as in the former case. Bronchial rhonchi dry and moist of various sizes and abundance are heard ; if the condensation be very dense, the bubbling rhonchi may acquire a ringing character. If excavations form, thin metallic echoing hollow rhonchus may be caught. The characters of the vocal resonance depend on the closeness and extent of consolidation ; null, weak or bronchophonic, but not snifflingly so, as in hepatisation, it may be whisperingly pectoriloquous over the site of a small deep-seated excavation with intervening condensed and infiltrated texture.†

1051. In the third case (*c*) of acute miliary tuberculisation, inspection, mensuration, and application of the hand, give results either completely negative, or those observed in acute bronchitis, —results more calculated, consequently, to mislead than to enlighten the observer. Neither does percussion furnish any trustworthy sign; the conditions of dulness are not only, absolutely speaking, very slight in amount, but they so equally pervade both lungs, that any slight defect of resonance discoverable would naturally be ascribed to individual peculiarity. At the outset the percussion-sound, just as in acute bronchitis, may be increased in clearness and raised in pitch. The respiration uneven, harsh, high-pitched, mingles here and there with dry bronchial rhonchi, or with sub-crepitation: vocal resonance gives no sign.

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* Hodson, U. C. H., Males, vol. ix., p. 16 : vocal fremitus increased in some spots, impaired in others.

† Hodson, U. C. H., loc. cit., case fatal in nine weeks.

The florid-coloured stage of pneumonia has no more positive signs than those set down previously; if hepatisation supervene, which is rare in this form of acute phthisis, its signs, in the main those of the simple disease, will want much of their characteristic definiteness.

1052. *Symptoms.*—The symptoms of acute phthisis in the forms *b* and *c*, are those of a febrile affection, with more or less positive functional implication of the lungs. The invasion, sometimes occurring in a state of apparent health, or preceded remotely by various depressing influences, such as anxiety of mind and over-toil, and immediately by exposure to cold and wet,* is marked by rigors, followed by acrid heat of skin; the rigors may recur on several successive days, and there may subsequently be perspirations with abundant crops of sudamina. I have seen one or two specks on the abdomen, having some, but by no means all, of the characters of the papular special eruption of typhoid, or Peyerian, fever:† and wholly doubt the correctness of an opinion ascribed (but not shared) by M. Leudet‡ to M. Waller, of Prague, that the true lenticular spots of that fever are to be found in some cases of acute phthisis. Petechiæ do not appear. Epistaxis, followed by coryza, may occur on the second day of seizure.* Prostration sets in early,—in a few days the patient may be unable to stand. Thirst; total anorexia; epigastric tenderness; dry lips and tongue; dental sordes; all signify digestive disturbance: but the form of the abdomen is natural, there is no gurgling in the iliac fossa; diarrhœa is rare; and constipation may be extreme, though there be abdominal pain and ulcerated intestine.† Restlessness, insomnia, cephalalgia, vertigo, tinnitus aurium, ‡ diurnal wandering and nocturnal delirium, bespeak cerebral sympathy.

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* Hodson, loc. cit.

† Garrett, U. C. H., Females, vol. vi., p. 146. May 3, 1851.

‡ Phthisie aiguë, Thèses de Paris. 1851.

Pain in the chest, variable in seat and never intense; cough (sometimes preceding, sometimes following, the fever in order of development) paroxysmal or not, and either absolutely dry or accompanied with expectoration of clear or yellowish and opalescent mucus, or in rare instances of viscid sputa, slightly stained with blood, without actual hæmoptysis; dyspnœa of considerable amount, indicated not only by the absolute frequency of breathing, but by perversion of its ratio to the circulation, and lividity of the face, constitute the chief of the thoracic symptoms.

The relationship of the pulse to the respiration, however, varies; the average, in my cases, has proved 3 : 1; but I have known it once fall to 1·54 : 1 (80 : 52).§ The heart and its membranes remain unaffected. The urine, of medium specific gravity, has the characters of febrile urine generally; it contains neither albumen nor sugar.||

1053. Acute phthisis occurs as the sole existing morbid state; or it destroys life suddenly in the course of chronic phthisis; or it forms the terminal phenomenon of certain slowly destructive maladies. In the latter remarkable aspect, acute phthisis has hitherto never been even glanced at, but I have within the last

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* Garrett, U. C. H., loc. cit.

† Hodson, U. C. H., loc. cit. Vide note, p. 510.

‡ Garrett, U. C. H., loc. cit.

§ Garrett, loc. cit. But there were here some hysterical symptoms; and the ratio soon changed to one of 3 : 1.

|| Garrett, loc. cit., pp. 150, 151.

year seen two most striking examples of the class. Two men, of middle age, dying of paraplegia, had myelitis, sharply limited to the dimensions of an oblong, horse-chesnut looking mass developed in the spinal meninges. In one of these cases the tumor had the microscopical constitution of tubercle:* in the other those of low exudation-matter.† In the former instance the patient died with symptoms clearly traceable to the acute tuberculisation found in the lungs; in the other instance the tuberculising process was latent. In both patients the anatomical characters of acute phthisis were perfectly developed. Tuberculisation may, then, imitate pneumonia, in bringing to a sudden close the career of lingering sufferers.

1054. *Diagnosis.*—Taking the symptoms now enumerated in connection with the physical signs of each form of acute phthisis, can the diagnosis of either be established?

1055. The softening form (*b*) may at first be diagnosticated through the intensity of the general symptoms, contrasting, as they do, strongly with the slight amount of pectoral disturbance,—the ordinary signs of pneumonia, intense bronchitis, and pleurisy, being absent; while general and increasing dulness under percussion, coupled with the signs of breaking up of tissue, eventually render error difficult. I say render error difficult, because double pneumonia running to suppurative destruction of tissue could alone, besides acute phthisis, produce such physical signs; and had such pneumonia existed, it would have been revealed at first by the signs of its early stage.‡ Acute cancerous infiltration of the lung may destroy life in less than four months, and simulate acute phthisis: but that disease is limited to one lung commonly, is attended with signs of tumor about the main bronchus, often with peculiar hæmoptysis, severe local pain, and diminished width of the side.

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* Anson, U. C. H., Males, vol. ix., p. 254.

† Knowles, U. C. H., Males, vol. x. p. 18.

‡ Pneumonia may, however, exist at first, furnish its own signs in more or less perfection, its rusty sputa, and perverted pulse-respiration ratio, and give place to acute tuberculisation. Hodson, U. C. H., loc. cit.

1056. The diagnosis of the miliary form (*c*), where grey granulations accumulate sparsely through the lung, with intervening arterial injection of tissue, though greatly more difficult, may commonly be effected by the following considerations. In idiopathic asphyxiating bronchitis the skin is warm only, or slightly cool and moist, its colour generally cyanotic or livid; bronchial rhonchi, dry and moist, are more abundant than in acute phthisis, and the moist class most abundant in the former disease inferiorly, in the latter often superiorly; the pulse-respiration ratio is less perverted in primary bronchitis than in acute phthisis; in the former full muco-purulent expectoration soon occurs, fails to appear in the latter. Neither the percussion-note nor the respiration-sounds afford positive help in the diagnosis. Again, pleurisy cannot be supposed to be present, for its physical signs are wanting. And if, for the first two or three days, the perverted ratio of the respiration and pulse might lead to the diagnosis of pneumonia (which, be it observed, exists in a certain state), the lapse of twenty-four hours will prove that the disease is not common idiopathic pneumonia,—for the signs of hepatisation are not an iota more obvious than the previous day.

1057. Either form of tuberculisation may be confounded with delirium tremens, simple meningitis, and typhoid fever.

1058. I once saw a patient after a week's illness (a hard drinker, who had frequently had attacks of delirium tremens), with moist tremulous tongue, foul breath, confined bowels, total anorexia, cephalalgia, excited unquiet countenance, general tremors, moist skin, and various delusions: he coughed, it is true, expectorated some muco-epithelial matter, and his breathing was hurried; but these symptoms were thrown into the shade completely by the class indicating a mild seizure of delirium tremens. However, the cerebral symptoms yielded in a few days; the pectoral grew more severe; and death took place in six weeks, under all the conditions of acute phthisis, the symptoms of which had at first been modified by the habits of inebriety of the individual. The patient had previously exhibited symptoms of phthisis, which had remained perfectly quiescent for at least twelve months.

1059. Where mingled excitement and adynamia are more than usually marked, acute phthisis might be mistaken for acute simple meningitis; but the following characters of the latter disease will be wanting,—vomiting at the outset, incoherence, violent delirium, wild glistening eye, photophobia, and intolerance of sound, strabismus, alternate flushing and pallor of the face, convulsions, and muscular rigidity or paralysis.

1060. The prostration and delirium of acute phthisis are scarcely marked enough to simulate those of pyohæmia at the outset; still, I have found it satisfactory to be enabled, by the history of the case, to exclude the latter affection from consideration.

1061. The chief difficulty in my experience (the point has already been touched upon by M. Louis) consists in distinguishing miliary phthisis from *typhoid* fever—fever of the Peyerian species. Dyspnœa, prostration, bronchitic rhonchi, duskiness of face, febrile action, dry skin, adynamic state of the tongue, delirium, and stupor, exist in both affections, and may do so to similar amounts. If pneumonia be present, it affords no help in the diagnosis; for it may be supposed secondary to the typhoid fever. The abdominal symptoms and the peculiar eruption of typhoid fever draw the line, to all appearance, positively,—but only in appearance: for abdominal symptoms and enlargement of the spleen may be present in acute phthisis, if the intestine be undergoing acute tuberculisation; and although eruption probably exists in all cases of typhoid fever, it certainly escapes detection, possibly from its slight amount, in a few instances. All these difficulties were well illustrated by a case, of which I subjoin the main facts:—

H. Manning, admitted U.C.H., Aug. 9, 1850, (Males, vol. v., p. 168), ætat. 23 (unable to give account of himself, subsequently known from friends to have been taken ill, July 19); prostration, stupor, dingy face, nails livid, skin warm, P. 120, R. 42, tongue dry and cracked, sordes on teeth, *spleen* 1½ hand's breadth high, tenderness in right iliac fossa, abdomen of medium prominence, and generally tender, *diarrhœa* for last two days; no sudamina; no distinct typhoid specks; general percussion-dulness at left back, with diffused blowing respiration; same signs less marked at right back; sputa viscid, somewhat transparent, of faint tobacco-juice tint; heart's size and sounds natural. *Diagnosis: Typhoid fever, with secondary pneumonia* (the difficulty about the want of eruption being noticed at the time). *Death*, Aug. 14, (the 28th day). There was double hepatisation mainly of the lower lobes; and the entire of both lungs were profusely studded with semi-transparent grey granulations (some growing opaque in the centre); a stratum of recent lymph in the left pleura was similarly studded; the spleen, 6 in. high, weighed 11 oz.; liver fatty; Peyer's patches contained crude yellow tubercles, here and there, size of pins' heads.

1062. The obscurity, thus arising from tuberculisation of the intestine, has not been referred to, so far as I know, by any author. But here is even a more difficult combination. A maid-of-all-work, aged twenty-one, about one year in London, is seen, on from the seventh to the tenth day of an acute attack, unable to stand, somewhat thinned, having, on the second day, had epistaxis (followed by coryza); with brown furred tongue, epigastric tenderness, slight vomiting, one or two doubtful typhoid specks on the abdomen, vertigo, tinnitus aurium, cough, with very little expectoration, slight limited dulness at both posterior bases, (P. 80, R. 52); and constant decumbency on the back. It is true, neither gurgling in the iliac fossa, diarrhœa, nor sudamina, existed; but the two first are often wanting in typhoid fever; and it was too soon to expect sudamina, on the hypothesis of typhoid fever. The pulse-respiration ratio was valueless, because the woman had hysterical hyperæsthesia of the skin. Yet this case shortly proved to be one of acute phthisis, running a peculiar course, to be by and by referred to; the meninges were free from granulations.*

1063. The *prognosis* of acute phthisis is of necessity almost absolutely fatal,—not, however, immediately so. Thus the patient, just spoken of, seized at the close of April, suddenly improved so much in July, that convalescence from the first attack must be admitted to have set in; she walked about the wards, and had scarcely any symptoms. In August another acute attack supervened, which cut her off on the tenth of October.

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* Garrett, U. C. H., Females, vol. vi., p. 145.

1064. The *treatment* of acute phthisis is far from being well understood,—the rarity with which the disease is diagnosticated explains this. M. Leudet, as matter of experience, lauds the expectant method.

The tendency to pneumonia justifies cautious use of the lancet, or, in doubtful cases, the local removal of blood by cupping. Counter-irritation at a certain distance from the chest will probably be serviceable: large blisters to the chest itself are injurious. Purgation must be avoided, from the danger of exciting the tuberculising process in the intestines. Diaphoretics and sedatives are advisable; and acetate of lead and digitalis have some claim to trial. The inclination to failure in strength, from the first almost, makes the propriety of exhibiting antimony doubtful; mercury seems a more hopeful medicine, and has certainly not yet been fairly tried. The acute symptoms having been successfully combated, the management of the disease becomes that of its chronic form.

SYPHILITIC DEPOSIT IN THE LUNG.

1065. The lung appears to hold a place among the textures in which the tertiary effects of the syphilitic poison exhibit themselves. Gummata, of the same anatomical constitution as the well-known subcutaneous product, have been described by MM. Ricord and M'Carthy as forming in the lungs, especially perhaps towards their periphery and bases. In the former position they look not unlike nodules of lobular pneumonia.

These gummata soften and are eliminated much in the manner of tubercle,—originally having, it is alleged, a consistence rivalling that of scirrhus: they are non-vascular.

1066. I can find no positive answer to the query,—do these gummata ever form independently of other tertiary evidences of syphilis in the bones and cellular tissue? If they do, their diagnosis must be infinitely difficult,—difficult indeed under all circumstances; for the physical signs must be those of solidification followed by softening and excavation, while the local and general symptoms closely simulate those of phthisis.

1067. There is strong motive to suppose that iodide of potassium is the essential remedy, and that mercury is contra-indicated: precise information is, however, wanting. Cod-liver oil should of course not be neglected.

CANCER OF THE LUNG.

1068. Cancer of the lung, most commonly of the encephaloid species, occurs in the forms of secondary nodule, and of primary infiltration, accompanied or not with tuberous formation in either mediastinum about the main, especially the right, bronchus.

1069. *Secondary nodules* may be perfectly *latent* in regard of symptoms; and if of small size, they are with difficulty detected by percussion, and may not produce pressure enough on the lung-substance to modify the respiratory sounds. Bronchitis occurs sometimes; but may be totally absent even where the nodules are sufficiently close and bulky to cause marked percussion-dulness. Dyspnœa is probably the only symptom distinctly traceable to these nodules.

1070. The physical signs of *primary infiltrated* cancer of the lung are numerous. The affected side is flattened, or even generally retracted, and the intercostal spaces slightly deepened; the respiration-play impaired; the vocal fremitus increased, if the infiltration be slight; diminished, or even annulled, if it be extensive, or, especially, if coupled with tumor non-adherent to the chest-wall; the heart's impulse may be felt, too distinctly, through the lung. The percussion-sound is dull, or hard, wooden, and even tubular, especially about the third rib; the dulness sometimes extends across the middle line. The respiration is of diffused blowing type, unless the main bronchus undergoes accidental closure from the pressure of a surrounding cancerous mass, when the respiration grows weaker and weaker, and may eventually be almost suppressed. Bronchophony sometimes exists markedly. Should softening and elimination of the infiltrated texture occur, cavernous respiration and rhonchus supervene, unless there be accidental closure by pressure of the main bronchus from superadded tumor. The diaphragm may be raised slightly on the affected side; the non-cancerous lung (for the disease is limited, in the great majority of cases, to one organ) gives signs significant of health, of hypertrophy, of emphysema, or of bronchitis.

1071. Here we have the signs of a disease diminishing the bulk of the lung, and causing retraction of the side. But this disease may be associated with actual cancerous tumor in the mediastinum, which, as will hereafter appear, has a dilating influence on the chest-walls. A combination of the signs of the two opposing states is met with in instances of their association.

1072. Primary cancer of the lung may, in infinitely rare cases, run a *latent* course. In the great majority of instances, manifest *symptoms* attend its progress. Pain, of variable duration, intensity, extent, and constancy, apparently occurring either in the cancerous lung itself, in the pleura, or in the intercostal nerves, is a very constant, and sometimes has proved the first, symptom. Dyspnœa, either slight, or troublesome, rarely acquires any great intensity, unless there be co-existent tumor. Cough, an invariable attendant, is, in very rare instances, dry; in the great majority, attended with expectoration. The sputa may be simply catarrhal, purulent, or bloody. In the latter case, the blood seems thoroughly mixed with serosity, mucus or muco-pus, and the sputa, commonly opaque, sometimes slightly translucent, are of the colour of red or black currant jelly (and not very unlike those substances), or pink. Encephaloid detritus has in rare instances been expectorated, of cognizable physical characters. The frequency of hæmoptysis has already [890] been referred to. Excessive fœtor of the breath or sputa, with or without obvious local gangrene of the lung, occasionally occurs.

Infiltrated cancer produces no centrifugal pressure-signs, and the only centripetal signs of the kind which it appears, unassisted by tumor, to have positively entailed, are dysphagia and obstructed breathing. If associated with mediastinal tumor, the various pressure-signs of that disease will be more or less prominently present.

1073. The general symptoms are sometimes slight. Emaciation, though slow in appearing, once established, advances rapidly. Night perspirations sometimes occur profusely; febrile action is not often highly marked; the pulse varies in characters. The skin commonly exhibits some shade of the straw-like or waxy cancerous tint. Increasing dyspnœa and cough, insomnia, failure of assimilative power, sometimes attended with anasarca of the lower extremities, (but this rarely occurs to any extent unless tumor be present,) put a term to existence.

1074. The mean duration of cancer of the lung, I formerly found, might be estimated at 13·2 months; the greatest 27 months; the least 3·5 months.

1075. *Diagnosis.*—Infiltrated cancer can only be confounded with diseases lessening the bulk of the lung.

1076. It is distinguished from *chronic pneumonia* by the notable amount of flattening of the side; by the occasional extension of dulness beyond the median line,—which never, so far as I know, occurs in *chronic* pneumonia; by any sign of centripetal pressure present, as dysphagia or weak respiration from bronchial pressure, for such never occur in pneumonia: by the progress of the disease, which, stationary in pneumonia, leads to excavation in cancer; by the peculiar jelly-like or cancerous expectoration in the latter disease, and by the much greater severity of its local symptoms.

1077. From *tubercle* cancer differs by the signs of extensive consolidation being unattended with rhonchus; the absolute limitation of the disease to one lung, which, common in cancer, is infinitely rare in tubercle; by any signs of centripetal pressure, as gradual suppression of respiration on the affected side; the jelly-like expectoration; the less severity of the constitutional symptoms, and the total absence of the secondary morbid states of phthisis. If the cancer have softened, the microscopical characters of that product may be found sometimes in the sputa.

1078. From *chronic pleurisy, with retraction*, cancer will be distinguished by the less amount of deepening and narrowing of the intercostal spaces; the greater respiratory play; the less irregularity of surface; the greater amount of respiratory sound, especially in the inferior regions; the natural position of the shoulder, scapula, and spine; the absence of friction-sound; by any centripetal pressure-sign that may be present; the peculiar expectoration; by the greater severity of the local symptoms as a whole; and by the history of the case, indicating in the one instance a disease on the increase, in the other on the decline. If *dilatation of the bronchi and "cirrhosis"* co-exist with chronic pleurisy, and if the bronchi be the seat of active secretion, while the constitution becomes implicated, the case will be less easily distinguished from cancer. But many of the points already enumerated will still avail; hæmoptysis, or the peculiar expectoration will probably have occurred, if the case be cancerous. The existence of external cancer may aid in the diagnosis in any of the above cases.

1079. Cancer seated in the lungs is even less under the control of remedies than when occupying some other localities. No evidence exists of the utility of arsenic or conium; cod-liver oil deserves a trial. Symptoms must be relieved as they occur, by ordinary measures. Dyspnœa is best mitigated by dry-cupping and blistering; small bleedings give temporary relief, but they cannot, of course, be often repeated.

ACEPHALOCYSTS.

1080. Acephalocysts, containing echinococci, form in the lungs, primarily:—secondarily, as far as can be ascertained, to similar entozoa in the liver, but without any direct connection with these,—and, lastly, are found in transitu outwards from a cyst seated in the liver. Under all these conditions acephalocysts may, or may not, be expectorated during life.

1081. The anatomical characters of the echinococcus, of the acephalocysts it inhabits, and of the mother-cyst, are the same in this organ as in their more common seat, the liver. The mother acephalocyst sometimes lies in direct contact with the pulmonary tissue, and is rarely surrounded with thickened cyst-like wall. The sac rarely attains great size, and commonly contains fluid of the same physical aspect as that of hepatic acephalocysts,—hence probably of the same very low specific gravity, slightly albuminous, and markedly saline, composition. Though occurring in all parts of one lung only, or of both lungs, acephalocysts exhibit a preference for the bases: in one remarkable case, the pulmonary veins afforded them a nidus.*

1082. Acephalocysts may be found in the midst of perfectly healthy tissue: this is very rare. Bronchitis, pneumonia, pleurisy, gangrene, perforation of the pleura and pneumothorax, singly or variously combined, have been met with as local effects. A case recorded by Laennec renders it highly probable that a pulmonary sac may make its way through the diaphragm, and discharge its contents through the intestines. In M. Andral's case, where the entozoa occupied the pulmonary veins, the right heart underwent dilated hypertrophy, probably from the obstruction in the venous circulation.

1083. Acephalocysts in the lungs may be the sole organic disease existing; or they may be associated with acephalocysts in the liver, mesentery, and spleen. In one certain instance they co-existed with tubercles in the lungs,—in another with cancer of the uterus,—in another with ascarides lumbricoides.

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* Andral, Clin. Méd. éd. 2, t. ii., p. 412.

1084. The clinical aspects of the disease differ so materially when the lung alone is implicated, and when perforation of that organ from an hepatic sac occurs, that they must be separately considered.

1085. I. *Acephalocysts limited to the lung, or at least not directly connected with an hepatic sac.*—There may be a total deficiency of subjective symptoms,—the disease, in fact, being *latent*, provided the cyst be of moderate size, and have not irritated the adjacent tissues. In a case of this sort Andral had found exaggerated respiration, inexplicable at the time, on the side where the sac existed.

1086. But the rule is that symptoms, essentially phthisical in character, occur. Cough, dyspnœa, more or less severe chest-pain, and inability to lie on one side; expectoration, catarrhal or bloody, followed by that of fragments of the walls, or of perfect acephalocysts, of various sizes, gives at length the special character to the disease. Night-sweats, but especially emaciation, may become extreme. The hydatid expectoration generally occurs paroxysmally, and may, on each occasion, be preceded by suffocative dyspnœa and great general anxiety: the quantity of entozoal substance voided at any one time varies from a few microscopical fragments up to a pint and more of unbroken acephalocysts. Neither bile nor urine is expectorated.

1087. Expectoration of acephalocysts may continue in such cases through a period of several months: generally serious pulmonary symptoms precede their first, and follow their final, elimination.

1088. So long as a sac remains unbroken, the physical signs simply indicate solidification; the quantity of respiration-sound will vary with the existence or absence of pressure on an important bronchus. If breakage take place, and the sac communicate with the bronchi, tubular or amphoric percussion-note, and the whole class of cavernous auscultatory sounds, will ensue on evacuation of the mother-cyst.

1089. The diagnosis from phthisis will in many cases probably prove impossible, unless acephalocysts be discoverable in the sputa; such discovery, too, will alone distinguish gangrene produced by this disease from gangrene of other kinds. From the observations of Dr. Peacock ("Edinburgh Journal," 1850), it may be expected that the microscope will prove of service in this way: the hooklets of the echinococcus may sometimes be distinctly recognized in the sputa,—probably, too, when the naked-eye evidences of the entozoa are deficient.

1090. Of twelve cases belonging, the majority certainly, the small minority very probably, to the present category, seven occurred in males of a mean age thirty-three,— five in females, of a mean age thirty-one. No clue is afforded by past experience to the ætiology of the affection.

1091. The prognosis is far from very unfavourable in this class of cases. In nine cases here were the results. Three ended fatally; but in one of these tubercles existed, and in another death was not even accelerated by the hydatids: the issue was doubtful in a fourth: while in the remaining five, perfect recovery took place. In every instance expectoration of hydatids had occurred.

1092. II. *Acephalocysts entering the lung from the liver, or, originally pulmonary, communicating secondarily with an hepatic cyst.*

1093. In cases of this class more or less obscure hepatic disturbance has been noticed for a variable period before the outbreak of severe symptoms indicating communication with the lung,—intermediate pleurisy sometimes occurring before the pulmonary tissue itself has been involved. The signs of hepatic disease have been epigastric pain, occasional nausea, and vomiting without obvious cause, failure of appetite, pains about the right infra-axillary and scapular regions, evidences of enlargement of the liver, and dingy earthy yellow or actually jaundiced skin, with icteric urine and fæces. But the real nature of the hepatic disease has, in recorded cases, scarcely even been suspected.

1094. When the lung becomes involved, very serious symptoms ensue. The patient is unable, from pain, to lie on the affected side; jactitation, pinched anxious countenance, and dread of death; jaundice of variable depth of hue; loaded tongue, sickness, vomiting of food and bile, anorexia, thirst, constipated and subsequently diarrhœal icteric stools; cough of paroxysmal character,—the paroxysms attended with lividity of face, cold extremities, clammy surface, dysphonia, and all the signs of semi-asphyxia, until relief is obtained by copious expectoration of acephalocysts,* more or less deeply bile-tinged, perfect or in detritus, mixed or not with mahogany-coloured fœtid fluid, sometimes even with cognizable shreds of sloughed lung; excessively frequent, small unsteady pulse, and fluttering palpitation of the heart; icteric urine; muttering delirium, nocturnal and diurnal,—all taken together make error in diagnosis impossible.

The physical signs are those of more or less extensive lung-consolidation, followed by those of excavation,—tubular, amphoric or cracked-metal percussion-note, hollow metallic cavernous respiration, pectoriloquous echo and thin metallic ringing rhonchus.

1095. An acute seizure of this character may prove fatal within a few days; or the patient may struggle through one or more attacks of subacute pneumonia, continue to expectorate hydatids from time to time for some weeks, and eventually recover. Permanent change of voice remained in one case recorded by M. Andral. But death has been the common sequel of such attacks; and the progress is greatly more unfavourable than when the lungs alone furnish the hydatids.

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* I have known nearly a hand-basin full of acephalocysts discharged in this way by an adult male, without immediately fatal results. The asphyxial state clearly depends on temporary obstruction of main bronchi, and is identical in nature with that observed in some cases of plastic bronchitis.

1096. Of nine cases, belonging to this class, two occurred in males of the mean age thirty-one at the time of observation; seven in females, whose age averaged thirty.

1097. The precise nature of the attack can of course only be diagnosticated by careful examination of the matters expectorated. Bile-stained hydatids, whole or in fragments, even microscopical particles, such as the hooklets, settle the question of nature and seat at once,—they exclude simple hepatic abscess, and perforating pyelitis.

1098. Of the specific *treatment* of pulmonary, as of other hydatids, little is known. Chloride of sodium and iodide of potassium, presumedly deleterious to hydatid life, should be tried. But admitting the vitality of existing echinococci to be destroyed, the elimination of the acephalocysts remains to be effected,—unless in the defunct state these entozoa cease to act as irritants to the lung. Natural cure of hydatids by accumulation of plaster-like material within the mother-cyst has not been observed, as far as I am aware, in the lung.

Should an external swelling appear, it seems advisable to open it. M. Freteau (Ann. de Montpelier, t. xi.), forty years ago, opened a sac in the right dorsal region; upwards of four hundred fragments of hydatids were discharged by the wound, and fifty expectorated: the patient, a male, recovered.

ENTOPHYTES.

1099. Some while since, Dr. H. Bennett figured an entophyte, allied in appearance to the Penicilium glaucum of Link, and found on the walls of tuberculous cavities: the jointed tubes of the entophyte had been expectorated two days, at least, before death.

1100. Virchow found an abundant crop of Sarcinæ Goodsirii in a gangrenous portion of lung: they must have grown where found. More recently, Zenker gave the particulars of a case where, though found in the lungs, the sarcinæ might, as a bare possibility, have made their way into the trachea before death from vomited matters, containing the entophyte in abundance.

The discovery has no practical significance at the present day.

SECTION VI.—DISEASES OF THE MEDIASTINA.

MEDIASTINAL TUMOR.

1101. I have seen in the mediastina tumors, composed of simple exudation-matter, scirrho-encephaloid, encephaloid, fibrous, and fibro-fatty substance: the local symptoms and the physical signs (inasmuch as both are mainly of mechanical origin) are almost identical, whatever be the constitution of the tumor.

1102. When a tumor occupies the mediastina, and encroaches, as it commonly does, mainly in front, bulging of the sternum and costal cartilages, in a variable spot and to a variable superficial extent, may exist; the intercostal spaces, widened and flattened, are then unaffected by respiration: but, on the other hand, no shadow of bulging may exist, and yet from other signs the presence of tumor be indisputable. Wherever a tumor, of any thickness, reaches the surface, vocal fremitus is annulled; fluctuation, simple or peripheric, is not to be detected; and a double impulse is sometimes transmitted from the heart, and may be felt both by observer and patient as a sort of inward succussion. If the tumor encroach pretty equally on both sides of the chest, there may be no alteration in their relative semicircular measurement; the respiration-play is found to be impaired, and the impairment may fall rather on expiratory retraction than inspiratory expansion. If by chance either main bronchus (and it is much more frequently the right that suffers) be seriously obstructed, the respiration-play will be relatively deficient on that side, independently of the influence of any general excess of solid mass within the right thorax. Should the tumor encroach notably on one side or the other, and be at the same time adherent to the wall of the chest, the space between the middle line and nipple will be lengthened on the same side. Wherever tumor reaches, or closely approaches, the surface, percussion will furnish an excessively dull short sound, with highly marked parietal resistance ; the superficial dimensions of the growth may thus be traced in the front or in the spinal regions behind. The resistance and resonance of the heart and liver commonly differ from those of tumors ; and hence, if the natural and morbid structures are in juxtaposition, their neighbouring edges may be defined by simple or by auscultatory percussion. Near the trachea, or more generally in front, or even in the back, the percussion note may be tubular, or even amphoric: I have known it amphoric over the lower half of the right back. The auscultatory signs vary widely. The respiration may be weak, almost to suppression over the morbid mass, or (from pressure on the main bronchus) over the side generally; or it may be of diffused or even tubular or hollow blowing type: these differences will depend on the precise relationships of the growth to the bronchial tubes and parietes ; as will the absence or presence of bronchophony or pectoriloquy of the loud form. The heart's sounds are conveyed through the solid mass with undue intensity, and if this press on the aorta or pulmonary artery, there may be systolic murmur, simulating intra-cardiac murmur, at the base. Bronchial dry and moist rhonchi may be heard. The position of the heart and of the wings, or of either wing, of the diaphragm, will depend wholly on the direction in which the tumor grows : they may be considerably displaced, or retain their natural positions, even where a large mass occupies the mediastinum.

1103. But the most distinctive and striking symptoms of mediastinal tumors arise mechanically from their *compressive* and *perforative* actions: both kinds of action may be centrifugal or centripetal.

1104. *Centrifugal pressure* acting on the general chest-wall, the clavicle, the sternum, the spinal column, or the diaphragm, may push these parts outwards or downwards; acting on the heart drives it to the opposite direction from that whence it plays; and bearing on the intercostal nerves, gives rise to neuralgic pains.

1105. *Centripetal pressure* may act on the heart, especially the right auricle; on the aorta, innominate, either subclavian, or pulmonary artery; on the vena cava, superior and inferior, the innominate veins, the pulmonary veins and the azygos; on the trachea, bronchi, and lung-substance; on the vagus, recurrent, phrenic nerves and pulmonary plexus; on the œsophagus; and on the thoracic duct.

1106. Centripetal pressure acting on the right auricle, diminishes its capacity, lessens the quantity of blood reaching the lungs, and adds in one form to the dyspnœa induced by other causes. Influencing the pulmonary artery, its effects are in essence the same, but obstruction of this vessel more effectually loads the entire venous system tributary to the right heart. Compression of the aorta gives rise to certain physical signs, especially arterial thrill and systolic (possibly even diastolic) murmur; but I have not actually known it carried far enough to obstruct the calibre of the vessel seriously. Influencing the innominate or either subclavian artery, it weakens the corresponding carotid and radial pulses. Pressure on the superior cava or innominate veins distends the internal and external jugular, subclavian, axillary, superficial thoracic and superior epigastric, facial, frontal, and even dorsal veins; and produces a tumid spongy fulness of the base of the neck, swollen livid discoloration of the face and lips (which look in extreme cases distended almost to bursting), and œdema of the face, arm, and affected side of the thorax or chest generally. The sinuses of the dura-mater become clogged, and heaviness, stupor or actual somnolency ensues; besides the venous congestion of the brain entails cephalalgia and failure of motor and sensory power in the limbs generally: but I have never seen, even in cases of venous distension, so great that rupture of the cervical and facial veins seemed likely to occur under the slightest extra-strain, any approach to epileptiform seizure. Encroaching on the inferior cava, pressure entails distension of the abdominal veins, congestion and enlargement of the liver, congestion of the kidneys and slight albuminuria, various gastric disturbances, anasarca of the lower limbs and abdominal walls, and eventually ascites. Obstructive pressure of the pulmonary veins leads to dyspnœa, hæmoptysis, œdema of the lungs, and hydrothorax. From the special tendency of tumors to form about the right bronchus, the vena azygos must frequently be pressed upon, and its obstruction must interfere with return of blood from the vertebral sinuses, and cause congestion of the cord; now such congestion was in all probability the source of torpor, formication, and feeble motor power in the lower extremities in a case of mediastinal tumor seen some time since with Dr. Bascome. Forcing the trachea backwards, or to either side, pressure induces stridulous breathing, and weakness of voice (traction of the recurrent nerve may have something to do with both these symptoms); its effects on the main bronchus have already been referred to, and sequential to these, exaggerated respiration may be established in the other lung; pressure on the lung-substance may force even the persistent air completely from all the texture concerned. Acting upon the vagus and phrenic nerves, and pulmonary plexus, it variously impedes and perverts respiration, and probably disturbs cardiac action; involving the recurrent nerves, it variously modifies the voice and obstructs inspiration. If the œsophagus suffer, dysphagia more or less obstinate will be the result; if the thoracic duct, unusually rapid emaciation. Formidable as this list of miseries seems, I have more than once seen them all (with the qualifications made) united in one and the same individual.

1107. *Centrifugal perforation* of the chest-wall or spinal column, not unfrequent from aneurism, is rare from tumor: I have never known of an example, except where the growth was cancerous.

1108. *Centripetal perforation* has in rare instances been observed in the lung, pulmonary artery, and œsophagus.

1109. Pressure also acts irritatively, producing pleurisy, active hydrothorax, bronchitis, local pneumonia, and laryngotracheitis.

1110. The symptoms of mediastinal tumor are pain, very variable in amount; dyspnœa; cough, with or without sanguineous expectoration, of the jelly-like kinds [1072], or catarrhal; actual hæmoptysis; inability to lie with the head low, and eventually complete orthopnœa, the sufferer sometimes, for weeks before death, never daring to go to bed, and never enjoying more than fitful dozes. Or the patient may, when in bed, habitually lean forwards or sidewards, with the head supported on the hand, the elbow bent, so as to throw the tumor off the trachea or main bronchus. I have known this posture assumed during sleep, the patient being unconscious of the change: * an instance of consensual action. The constitution may long bear up against the local disease; but the dyspnœa and insomnia at length affect the appetite, and emaciation sets in. The patient dies gradually, anasarcous and exhausted, or he may perish suddenly from obstruction or perforation of the pulmonary artery.

1111. *Diagnosis.*—An intra-thoracic tumor may, according to its seat, be confounded with extensive chronic pneumonia and chronic pleuritic effusion, with chronic pericardial effusion, great enlargement of the heart, or aneurism of the thoracic aorta. The distinctive marks of the cardiac and arterial affections will be found in the descriptions of these.

Tumor will be distinguished from chronic pneumonia by the tendency to increase, instead of diminution, of bulk of the affected side, by the implication of the mediastinum, by the greater intensity of the dulness under percussion, the failure or the disappearance of vocal fremitus, which remains in chronic pneumonia, and the different characters of the respiration in the two diseases. Hæmoptysis and red jelly-like expectoration never occur in chronic pneumonia; whereas the emaciation is of earlier appearance, and more marked than in cases of tumor.

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* Holmes, U. C. H., Females, vol. vii., p. 120.

From empyema, tumor will be distinguished by the absence of intercostal fluctuation, simple or peripheric; by the greater intensity of dulness under percussion; by the limits of the dulness being either not at all, or very slightly, changed by altering the patient's position; by the interspaces not being convex; occasionally by the intensity of blowing respiration; by the clear transmission of the heart's sounds; by the comparative irregularity of outline of the side, some spots being more prominent than others; by the fact, that in cases of tumor, careful percussion will almost surely detect some spot giving a comparatively clear sound, where, according to the laws of physics, liquid, had this been the cause of the general dulness, must have made its way, and hence caused dulness there as well as elsewhere; by the whole class of centripetal pressure-signs; and, if they have occurred, by hæmoptysis, or by the jelly-like expectoration.

A patient having mediastinal tumor may also have empyema on the side most encroached on, generally the right. If the patient be seen for the first time, when the two diseases are present, and if precise medical information as to the previous course of the complaint be wanting, this combination may be very difficult of diagnosis. But empyema does not produce centripetal pressure-signs, nor give rise to hæmoptysis or jelly-like expectoration; if these symptoms be present, there must be, in addition to empyema, either tumor or aneurism. Besides the test of movableness of dull sound may be appealed to, though not with absolute confidence [139].

1112. Given an intra-thoracic tumor, how may its *nature* be determined? If the signs of infiltration of the lung co-exist, the tumor, as far as I have seen, is either composed of simple exudation-matter (of these I have examined three microscopically) or cancer. If the tumor present externally, it is cancer; if tumors exist elsewhere, either secondary to, or independent of, that in the chest, supposing even *their* nature cannot be established directly, the chances are strong, that the thoracic growth is cancerous. Violent hæmoptysis is more common with cancerous than other growths; expectoration of cancer can, of course, only occur with the former. The constitutional characters of cancerous disease may be wanting.

1113. Intra-thoracic tumor is, of course, beyond the permanent influence of *treatment.* But it is astonishing what marked temporary improvement of all the direct symptoms may be effected by cautious cupping, dry-cupping, flying blisters, profuse inunction with an ioduretted liniment, gentle purgation, and diuretics. I have twice, within the last year, known the diagnosis of intra-thoracic tumor contested, on the grounds of the relief produced by such measures; yet, *post-mortem* examinations, in one instance in three, in the other, in seven weeks, proved the existence of massy growths. Unfortunately, all therapeutical means soon cease to avail us; and I know no more truly painful spectacle than that of the closing sufferings inflicted by mediastinal tumors.

MEDIASTINAL ABSCESS.

1114. *Anterior Mediastinum.*—Abscess in the anterior mediastinum, a rare malady, is almost always of tuberculous nature,—and associated generally with strumous softening and abscess of the cervical glands.

1115. The physical signs are, in nature, the same as those of tumor: but in degree they differ. Inasmuch as fluids press equally in all directions, there will be proportionally less of the effects of centripetal pressure in the case of abscess than of tumor; the trachea, veins, and œsophagus will longer escape. The percussion-note, too, will not be so absolutely dull: thus in the case of a man with suppurating cervical glands, and all the physical signs of non-aneurismal, solid, tumor behind the first bone of the sternum, the amount of dulness very perceptibly decreased during three months' observation previous to death.* Inexplicable during life, this change was accounted for by the purulent liquefaction of a portion of a tuberculous mass in the mediastinum: fluid would of course make its way from the sternal surface, backwards and sidewards, in the direction of gravity, and permit the lung to come more forwards. It would, however, be a bold proceeding to diagnosticate liquefaction of a tumor on such evidence alone.

1116. Suppurative action is generally very painful in this situation; irritation of the pleura and intercostal terminal twigs combines with the pain of the cellular or glandular inflammation to wear out the patient.

1117. Generous diet, cod-liver oil, moderate local depletion and counter-irritation are the therapeutical means indicated.

1118. *Posterior Mediastinum.*—Abscess in the posterior mediastinum occurs sometimes from suppurative softening of bronchial glands; sometimes from tuberculous caries of the dorsal spine. The signs of the former will be found in Section III.; of the latter I know nothing by experience.

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* Buckley, U. C. H., Males, vol. ix., pp. 248—310.

CHAPTER II.

DISEASES OF THE HEART.

FUNCTIONAL DISEASES.

1119. Functional disturbance of the heart is indicated by increased action (*palpitation*), perverted action (*fluttering, or irregularity of rhythm*), *infrequent*, so called, *slow*, action, and action enfeebled more or less, even to extinction (*syncope*). These conditions, variously combined, may attend textural changes in the organ; but they occasionally exist, independently of any such appreciable change.

1120. The distinction of functional from organic disturbance of the heart is often far from easy. Many of the general rules given for this purpose fail clinically. The inconstancy of the symptoms of functional, and constancy of those of organic, ailment, are strongly dwelt on, for example; but all the subjective, and many of the objective, symptoms may disappear temporarily in cases even of extensive organic disease. The existence of secondary changes, such as subcutaneous œdema, and congestion of the lung, commonly proves the cardiac affection to be organic: but not always, for spanæmia, added to nervous palpitation, may induce œdema. If exercise relieves a disturbed heart, its affection is pronounced to be dynamic only: incorrectly, for, if spanæmia exist, exercise may be unbearable. If, in the intervals of attacks of disturbed action, the force and rhythm of the pulse and heart are natural, those attacks are said of necessity to be functional: an error; for the most perfect tranquillity of the organ *may* exist, from time to time, though its texture is seriously unsound. Careful and repeated physical examination alone will, in difficult cases, justify a positive opinion on this question. And the student must remember that the mere detection of a morbid physical sign in a palpitating heart does not justify the assumption of organic disease: for instance, a basic systolic murmur may be simply anæmic; a systolic murmur at the mitral apex may be generated by irregular action of the musculi papillares; and extended dulness to the right of the sternum may depend on temporary distension of the right cavities with blood. On the other hand the total absence of physical signs does not prove the heart to be in a perfect state of organic soundness: there are slight amounts of change in the heart's substance, of which the perverted signs (for, doubtless, such really exist) are beyond the penetration of the present day. Besides conditions of adjacent structures may throw physical signs into the shade: emphysema of the lung will prevent increase of percussion-dulness by an enlarged heart.

1121. Singularly enough, the amount of local suffering entailed by disturbance wholly, or in the main, functional is often greater—is, in the mass of cases, greater—than that produced by actual organic disease.

1122. Functional disturbance of the heart is connected more or less constantly and markedly with the following conditions: perverted innervation, as in cases of hysteria, spinal irritation, uterine and ovarian excitement, and intercostal neuralgia: altered condition of the blood, as in hæmorrhage, anæmia, gout, chronic rheumatism, and chronic disease of the liver; mechanical interference with the organ, as when the stomach or intestines are distended with food or flatus; certain poisonous influences, as that of strong green tea, and of various diffusible stimulants in excess.

1123. *Palpitation.*—In dynamic palpitation, action may be increased very triflingly or sufficiently to shake the entire trunk; the frequency of the systoles, commonly augmented, is sometimes greatly so; their rhythm may be regular or irregular.

1124. The impulse is too extensively visible, but the apex-beat natural in site.. If the heart be a well-nourished one, the impression it gives to the hand laid on the cardiac region, is that of a *blow* (the impulse may even be somewhat heaving); if a feeble organ, the impression is that of a *slap.* Systolic basic thrill may sometimes be felt [365]. Habitually, the area of dulness remains unaltered, but extension to the right of the sternum may occur, in prolonged paroxysms especially; dulness is never carried upwards. The first sound is too loud and clear at the mitral apex, and somewhat abrupt and short; the second is duller and less clicking than natural at the mid-sternal base. The first sound may be loud enough to be audible, both to the patient and to bystanders, at a distance of some inches from the chest. Reduplication of the second sound at the base is common; and a clear metallic ring, or a pericardial rub, may accompany the shock at the mitral apex.

1125. Will palpitation produce murmur? If there be the slightest co-existent spanæmia, basic systolic murmur will, of course, be generated; but this murmur may also occur, both in males and females, during violent palpitation, where no evidence exists of any morbid state of the blood. I suspect that mere palpitation may also cause passing mitral regurgitant murmur of dynamic mechanism [442]; but of this I am not sure.*

1126. The aorta, the carotids, and the arteries generally, beat with undue force, sometimes exceeding that of the heart itself;

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* In the recent valuable work of Dr. Stokes (which I did not see until the first part of the present volume had passed into the printer's hands), an opinion is by inference ascribed to me on the subject of inorganic cardiac murmurs, differing from that I have really professed. Dr. Stokes seems to infer that I deny the possible production of systolic murmur at the apex, independently of textural disease of the mitral orifice (Diseases of the Heart, p. 496). Far from this, I have always strongly insisted on the probability that *dynamic* murmur of this site and rhythm really occurs; but cardiac murmur of *blood-origin* I have supposed, and still suppose, to be always basic in seat. I have never yet heard in a purely chlorotic woman a murmur having all the characters of a mitral regurgitant one.

the pulse is quick and sudden, or full, hammering, and heavy, in some plethoric people, whose hearts are free from hypertrophy.

1127. Among the subjective symptoms may be enumerated choking sensations; a feel as if the heart were jumping into the throat, and the eyes bursting from the orbits, præcordial anxiety, faintness, actual syncope or partial insensibility; præcordial pain, slight, dull, aching soreness, or agonizing pangs, simulating angina, and sometimes relievable by pressure of the ribs, though the patient cannot lie on the cardiac side; hurry of respiration, sometimes out of proportion with the pulse, giving the sufferer the appearance of a person out of breath with running; tinnitus aurium; vertigo and confused vision; cephalalgia; heat of head and flushed face; and clammy coldness of the extremities. In severe attacks there is often extreme general distress, and fear of death,—singularly enough, more of this, often, than in cases of palpitation of organic origin: the consciousness of local suffering is rather in the ratio of the general and local sensibility, than of actual palpitation. Lasting for a few minutes, an hour, or with remissions for days together, a fit of palpitation frequently terminates by sleep.

1128. In the *treatment* of the paroxysm, the first effort must be to remove, or lessen the intensity, if possible, of its cause. The fit may be shortened by antispasmodics, asafœtida, musk, and valerian, especially in hysterical persons; by diffusible stimulants, ammonia, the æthers, and very strong coffee; by narcotics and sedatives, opium, hyoscyamus, hydrocyanic acid: if the excitement of the organ be great, and its action not distinctly irregular, digitalis may be used with propriety. Acidity and flatulence, frequent causes of palpitation, may be corrected by soda and cajuput oil; a loaded stomach freed by an emetic. If plethora of the sthenic kind be present, cautious venesection is advisable: if of the asthenic, digitalis tranquillises the organ speedily. Should a gouty or rheumatic state be discoverable, colchicum, guaiacum, and ammonia, and irritant applications to the joints, are the best remedies. Heat may be applied to the extremities; but the application of ice over the heart is a dangerous practice, especially if the rhythm of the organ be affected.

1129. Persons subject to palpitation should avoid stimulants, over-exercise, over-sleep, emotional and intellectual excitement. Hydrocyanic acid, aconite, digitalis, and belladonna, internally, varied according to circumstances (the latter also in the form of plaster over the heart), coupled with the use of the shower-bath, attention to diet, regularity of bowels, cheerful occupation, and, lastly, change of air, will either remove the tendency to palpitation altogether, or greatly mitigate the severity of its seizures. If there be the least spanæmia, iron is indispensable.

1130. *Perverted or irregular action* affects rhythm mainly, force secondarily. The varieties of perversion, as indicated by the heart's impulse, by its sounds, and by the relationship of these sounds to the arterial pulses, have already been described [361, 422].

1131. A heart beating, now fifty, now one hundred and eighty or more times in a minute, now with excessive force, the next moment with such feebleness that the hand scarcely catches the impulse, cannot fail to become more or less clogged and obstructed internally. The evidence of this appears not only in the syncopal and suffocative tendencies of such fits, when at all prolonged, but in the increased area of the heart's dulness,—especially to the right of the sternum. The quality of the heart's sounds changes from moment to moment,—the general tendency of the first of the two, especially, is to shortness and undue clearness; but occasional forcible contractions may give a healthy systolic sound, masked possibly by parietal shock.

1132. In this form of palpitation, præcordial anxiety generally reaches its maximum. The prognosis is more serious than in the previous variety. Although irregularity of action may exist in the very highest degree without valvular disease, I confess I have very rarely met with it under such circumstances, where there was not either certainty or strong suspicion of alteration of the heart's texture,—in the form of softening or fatty degeneration. At all events, I am persuaded that when elderly persons are the frequent subjects of what is called "stomach-palpitation," the assumption that, because no valvular signs are discoverable, the heart is sound, is a positive error. Excessive irregularity of the heart's action must always be ooked on as a serious affair; though exceptional individuals xist, who, by some peculiar idiosyncracy, enjoy good health, vith an habitually more or less irregular pulse: in these persons, t is to be remembered, too, the organ is quiet in action, though norbid in rhythm.

1133. During a fit of this form of palpitation, the stimulant nd anti-spasmodic plan of treatment is required. In the interals the tone of the heart may be improved by ferruginous nd other tonics, the use of the shower-bath, and removal to a racing climate.

1134. *Decreased action*, essentially affecting force, but also requency, if carried to extremes, produces *syncope;* and in its inor degrees, and if an habitual state, entails general languor f all the functions.

1135. (*a*.) Actual syncope, whether induced directly by ilure of the heart's irritability, or indirectly through deficient upply or perversion of nervous influence, or by loss of blood, ccurs with very much the same train of symptoms.

1136. Commonly some premonitory symptoms are noticed: ausea, sinking feel at the epigastrium, disturbed vision, ertigo, tinnitus aurium, confusion of thought, pallor, drawing f the features, inclination to clammy perspiration, tremulous ntractions of the muscles, or slight convulsions, chattering f the teeth, and failure of the pulse, announce the coming ncope. In less usual cases the actual stoppage of the heart's tion is sudden.

1137. In the state of complete syncope the pulse, though absolutely wanting at the wrist, may often be faintly felt in the carotids; the patient is totally unconscious; the surface cool, clammy, or natural; the features contracted; the nares pinched; the lips of marble pallor; the face and skin generally more or less blanched; the respiration suspended absolutely, or almost imperceptible: in some few instances the sphincters of the bladder and rectum relax. The heart's impulse may be almost or completely lost to the eye and hand; the sounds are rarely totally inaudible in ordinary syncope: both are, of course, exceedingly feeble, the first very short, the second generally lost at the apex.

1138. Lasting for an instant only, for seconds or minutes, ordinary syncope terminates by gasping or rather sighing respirations, at long intervals, and gradual return of pulse, consciousness, and colour. Sometimes vomiting or discharge of flatus, convulsions, palpitation, or profuse perspiration, take place at the time of returning consciousness. The sensations are, on the whole, painful and distressing.

1139. Syncopal unconsciousness will be distinguished from that of asphyxia by the pallor of surface; in the latter, congestion and general lividity of the head and face exist, and the heart continues, though feebly, to beat.—In uræmic coma, or semi-coma, there is a pallor of face to deceive, but it is an œdematous pallor; the odor of the breath is urinous; and, finally, the state of the heart's sounds and the fulness of the pulse at the wrist will prevent error.—Apoplexy, though traced *post mortem* to cerebral hæmorrhage, sometimes occurs with pallor of face; the state of the pulse and of the heart prevents the possibility of error,—in apoplexy, neither heart nor pulse fails in strength, and both may act with undue energy.—Hysterical insensibility, with absolute motionlessness, closure of the eyes, almost complete suppression of respiration, simulates syncope; but there is no pallor, and the pulse beats steadily and with distinctness.—Lastly, cases of prolonged syncopal trance occur where the pulse is imperceptible, respiration suspended, consciousness gone; and this state continues for several days: such a condition might easily be, and has been, mistaken for actual death. But in prolonged syncope, the countenance retains some life-like expression, and the thermometer marks a higher degree in the rectum and mouth than on the surface generally; besides, neither cadaveric congestions nor rigidity make their appearance; and if the skin be burned to the second degree over a small surface, a bleb will form, if the patient be alive, none if he be dead. Still in doubtful cases, before a positive opinion is hazarded, sufficient time should be allowed for putrefactive changes; lest a catastrophe, the most horrible the mind can conceive, occur. Never having observed a case of such apparent death, I know not whether the heart's sounds are absolutely annulled: Dr. Hope imagines the second might be heard, although the first were inaudible.

1140. In *treating* an attack of syncope, the first points are to place the patient horizontally with the head on, or below, the level of the shoulders, to allow a free circulation of cool air, and remove all pressure from the neck and chest. If the syncope be caused by loss of blood, a tourniquet may be applied with advantage to one or both femoral arteries. Stimulant impressions on the nerves,—on those of the nostrils and lungs by ammonia, strong acetic acid, the fumes of burning feathers,—on those of the skin by the cold water dash, the application of vinegar to the temples, slapping the palms of the hands or surface generally, frictions with stimulant liniments along the spine,—on those of the stomach, (if the patient can swallow) by a draught of cold water, frequently arouse the heart instantaneously. Æther, aromatic spirits of ammonia, or brandy, should be given internally, if possible, by the mouth; if this be impossible, and the fit be prolonged, enemata, with ammonia, turpentine, or brandy, may be administered. It is scarcely necessary to say, that if protracted syncope depend on an overloaded state of the stomach, an emetic should be given (by the rectum, if otherwise impossible): flatulence may be relieved by the rectum-tube, and an asafœtida and cajuput enema.

In cases of protracted fainting, assuming a serious character, hot applications, sinapisms or turpentine fomentations, to the heart and spine, electro-galvanism, and artificial respiration, must be successively had recourse to. If the cause be loss of blood, transfusion presents itself as a final measure.

1141. (*b.*) Where the heart is habitually feeble in action, coldness and clamminess of the extremities, œdema of the ankles and insteps, shortness of breath, frequent inclination to faintness, sensations of languor and ennui; low spirits, anorexia or depraved appetite, foul breath, and constipated bowels, are more or less constant symptoms. The heart's impulse is feeble, its sounds wanting in tone, reduplication of the diastolic sound at the base common, palpitation easily excited.

1142. This state of things, which I have principally seen in young females, and often in connection with disordered menstruation, is curable by attention to the state of the uterus, and by the tonic invigorating plan of treatment. Moderate walking exercise is essential.

1143. *Infrequent Action.**—Infrequent systolic action may be attended with quick and abrupt or with slow and drawling ventricular action; and the pulse may, or may not, accurately reproduce the number and the qualities of the ventricular action [424].

1144. Infrequent action seems in some instances a physiological peculiarity, and is probably congenital, existing without cardiac, cerebral, or respiratory disturbance: in such cases febrile disease increases the frequency and strength of the action, and lessens its intermittent tendency. Such a state may be wholly unconnected with cardiac textural disease of any kind.†

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* Infrequent action is commonly confounded with slow action: but in point of fact the action may be at once infrequent and quick, or at once frequent and slow.

† Vide an interesting case by Dr. Macdonnell, in Brit. Amer. Journal of Medicine.

1145. Infrequent action attends a certain share of cases of fatty change of the heart: each action is then quick. Each contraction, long in coming, is brief in duration: possibly the extent to which true contractile tissue is destroyed, renders the organ dilatory in responding to the natural amount of nervous influence exciting contraction. Irregularity of force and rhythm is the rule in these cases.

1146. In certain cerebral affections the ventricular action, lessened in frequency, is besides slackened in speed: the pulse is infrequent and slow. Here nervous influence is imperfectly and laggingly supplied to the heart, it may fairly be assumed; hence the rarity of contractions and prolongation of each.

1147. Infrequent action of cerebro-spinal origin is strong; of cardiac origin weak: in either variety the heart may not beat more than from twenty to forty times in the minute.

ANGINA PECTORIS.

1148. Angina pectoris is a paroxysmal neurotic disease, of undetermined nature, in which the heart is essentially concerned.

1149. *Symptoms.*—Pain, often accompanied with tenderness, in the lower sternal part of the præcordial region, shooting to the back, the left shoulder, and neck, and along the left arm to the fingers, or stopping short at the bend of the elbow, or extending to both arms, or much more rarely to the right arm only, sometimes passing to the left leg, or invading the four extremities at once, and, in rare instances, producing numbness in the testes,—præcordial pain of these sympathetic attributes, and perfectly sudden in its onset, is the essential symptom of a fit of angina. Dull, aching in character, lancinating, tearing, or indescribable,—an exquisite torture, constrictive, and suffocative, it produces, or is certainly coupled with, in the majority of sufferers, pallor, intense anxiety of countenance, and dread of impending dissolution. The heart palpitates with variable strength and rhythm; the latter, though sometimes perfectly regular, may be so disturbed as to produce tendency to syncope: the sounds and palpable impulse vary accordingly. If there be prominently-developed murmurs, there is certainly—if even the slightest murmur, there is probably, something more than mere anginal disturbance to produce them. The pulse, as noticed by Heberden, is not necessarily increased in frequency: it may be strong, full, and regular in rhythm (but this, hardly, except in mild seizures), or small, irregular, feeble and frequent.

Respiration is *secondarily* affected; there may be slight dyspnœa or orthopnœa, with lividity of the face,—yet, by an effort of the will (if the patient dares to encounter the pang this commonly, though by no means always, produces), the chest may be pretty freely expanded, and the breathing relieved for a brief space: dyspnœa, subjective or objective, is not a primary, or essential, phenomenon of angina.

Slight convulsive actions may occur; but, on the whole, the spinal and encephalic functions remain unaffected. There may be a full discharge of pale urine.

1150. Lasting a few minutes, or prolonged through an hour with changing severity, the fit goes as suddenly as it came, leaving, as its sequence, exhaustion, accompanied with strong sense of present relief; or death takes place by syncope.

1151. The intimate nature of angina pectoris seems scarcely susceptible of positive and direct determination. That the attack is essentially neurotic, appears from its sudden advent and departure,—from the character and intensity of its suffering, —from the perfect ease enjoyed in the intervals of seizures,— and from the kind of treatment that proves beneficial. The vagus and the sympathetic filaments distributed to the heart are doubtless the nerves implicated.

Pain is obviously one portion of the neurotic disturbance present. What other is associated with it? Heberden and Dr. Latham espouse the doctrine of cardiac spasm: admitting its existence, whether the pain is mainly, as is ably argued by Dr. Latham, the product of the spasm, or in greatest measure an independent neuralgia, causing the spasm, would still remain an unsettled point. But the presence of spasm at all is matter of doubt. To those reasoners who deny altogether the possibility of such a state as spasm of the heart, much attention need not be accorded: the time is not long passed when the possibility of spasm of the urethra was denied by the schoolmen. But Parry and Dr. Stokes, on the ground of observation, look upon temporary increase of weakness in an already weakened organ, as the essential muscular element of the anginal paroxysm. And I must confess that what I have seen of the complaint accords with this doctrine: at least I have never met with true angina in the possessor of a strong hypertrophous heart.

1152. There is not a single structural disease of the heart, of its nutrient arteries, and of the aorta, which has not been recorded, as present in different victims of angina pectoris. It is, on the other hand, affirmed that death may be produced by angina, the heart and vessels being texturally sound. I doubt this exceedingly: recent narratives invariably describe some organic change, and older accounts are not trustworthy, seeing that accurate knowledge of dilatation, softening, and various other morbid states is the acquisition of the last few years only. It has occurred to me to examine during life some six or eight cases of true angina; in every one there were signs of organic disease,—generally of flabby dilatation: I have opened, or seen opened, the bodies of three persons destroyed in the paroxysm; the heart was texturally affected in all. But, on the other hand, as angina occurs with all varieties of heart-disease, and may be absent with any one of the whole series, except perhaps very extensive calcification of both coronary arteries, the conclusion is unavoidable, that there is something of neurotic character beyond organic mischief, concerned in generating the paroxysms.

1153. That the disease is peculiarly rare among females, scarcely observed before the fiftieth year of age, and comparatively unknown among the humbler classes, are facts in its history inexplicable upon any hitherto broached theory of angina.* Laennec supposed that epidemic influence renders the disease unusually common from time to time.

1154. *Treatment.*—Sedatives and stimulant anti-spasmodics are the medicines essentially to be trusted to during the fit. The dose of opium will be measured by the intensity of the pain; from forty to sixty drops of laudanum or of the liquor opii sedativus may, in a severe case, be given along with brandy or from half a drachm to a drachm of æther or aromatic spirits of ammonia, and repeated according to the urgency of the suffering. Musk, camphor, and belladonna are of very inferior importance. Mustard poultices may at the same time be applied to the heart and to the dorsal spine, or cloths wetted with the strong liquor of ammonia laid upon the præcordial surface. Laennec's suggestion of the transmission of a magnetic current (with or without acupuncturation) through the chest, has scarcely been fairly tested. An electro-galvanic current, however, affords better chance of successful influence, and in a serious case deserves trial. The mustard pediluvium, especially if the patient be gouty, is useful; the application of chloroform to the præcordial region hazardous.

1155. Speciality in the circumstances of the attack may call for special treatment. If the patient be the subject of undoubted sthenic plethora, and especially if the heart be known by previous examinations to be a well-nourished one, the abstraction of blood from a vein, or by cupping between the shoulder-blades, is clearly indicated; but bleeding must not be heedlessly undertaken and without assurance as positive as is attainable, that the heart is at least not a dilated, soft, or flabby one. If flatulence and acidity exist, soda, cajuput oil, and sesquicarbonate of ammonia may be administered with the opiate medicines; if a large undigested meal lie in the stomach, it should at once (unless the breathing be very seriously embarrassed) be removed by an emetic of sulphate of zinc.

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* Of 88 cases, collected by Sir John Forbes, 80 refer to males; and of 84 patients, 72 had passed the fiftieth year.

1156. Angina of malarious origin, and recurring periodically, is said to be observed. In such cases the treatment would be that of miasmatic diseases generally.

1157. A person who has had one attack of angina pectoris must remember, that instances, in which recurrence does not take place, are altogether exceptional, and further, that the periods of recurrence gradually approximate more and more, and each successive paroxysm, as a rule, exceeds its predecessor in severity. A first attack is generally brought on by an effort of some kind, such as walking up a hill, or in the teeth of a sharp wind; but eventually the most trivial influence will suffice to produce a paroxysm; emotion of any kind, sudden movements of the trunk or arms, efforts at defecation, the acts of coughing, drinking rapidly, &c. Hence it is clear that the subject of angina must live according to the most stringent rules; every conceivable precaution must be taken to keep the heart in a tranquil state. The patient should give up exciting pursuits of all kinds,—intellectual, corporeal, and emotional,—and learn, if he be of the *genus irritabile*, to govern his temper. Daily exercise should be slowly taken on perfectly level ground, either on foot or in an easy carriage; riding on horseback is scarcely to be permitted with safety. The diet should be moderate in quantity, simple in quality; the bowels never allowed to be confined. A belladonna plaster worn over the heart, and an issue, seton, or perpetual blister to the arm, have appeared useful in some cases: if the patient have confidence in counter-irritation, this should by no means be neglected. Change of scene and travel, coupled with the use of tonics, vegetable or mineral, will, by improving the general health, render the patient less prone to seizures. Arsenic, nitrate of silver, and sulphate of zinc are the best of the class of mineral tonics, unless anæmia be present, when, of course, iron is *the* remedy. The removal of gout, chronic rheumatism, or old-standing skin-diseases should be very cautiously, if at all, attempted, in the subject of angina: relief of those complaints is unquestionably sometimes followed by increased severity of the cardiac affection.

1158. A paroxysm, of which the too experienced patient learns by his feelings to expect the approach, may sometimes be averted completely by an opiate; and sufferers should always carry, properly protected, on their persons, an antispasmodic and sedative draught. One of the best safeguards against seizure consists in the idea, that the means of averting evil consequences, if it occur, are within reach.

PSEUDO-ANGINA PECTORIS.

1159. Angina pectoris is a rare affection; there exists, on the contrary, a complaint simulating many of its characters, which is of considerable frequency, and may be termed pseudo-angina.

1160. The symptoms are, more or less severe, commonly constrictive, pain referred to the region of the heart, palpitation occurring paroxysmally without obvious cause, or under exertion, or through over-eating, indigestion, flatulent distention of the stomach, and a variety of other functional disturbances. The breathing becomes panting and suspirious. Giddiness and faintness are sometimes observed. The patient dreads efforts of all kinds.

1161. Physically the heart may be to all seeming healthy. The apex beats in the usual spot, and with natural characters; even in the height of the paroxysm no murmur of any kind exists. Shortness, feebleness, and clearness of the first sound at the mitral apex are the only conditions at all frequently met with. These indicate tendency to dilatation, and weakened power: the impulse sometimes wavers and is of uneven force from time to time. But even in cases, where all seems physically normal, there may be organic disease beyond the diagnostic means of the present day: if so, however, it must certainly be slight.

1162. Like true angina its imitation is more common (though by no means to the same extent) in males than females, and in the higher than in the lower walks of society; but, unlike true angina, frequently occurs in young adults.

1163. Pseudo-angina has fallen under my notice as an attendant on various hæmic diseases,—chronic gout and rheumatism, and spanæmia; on nervous diseases,—hysteria, spinal irritation, and epilepsy; on intercostal and mammary neuralgia, the direct evidences of which are more or less complete. Pseudo-angina also occurs in connection with a variety of nervous disturbances, the influence of which can only be made intelligible by reference to some physiological facts. That *suddenly* perverted dynamism of the brain and cord affect the heart's action is proved by the phenomena of simple concussion of those centres; the influence of irritation of the roots of the spinal accessory, of the first four cervical nerves, of the par vagum, of the phrenic and some intercostals, of the cervical ganglia, especially the first, of the cardiac nerve, of the semilunar ganglia and solar plexus, of the nerves of the stomach, of the hepatic, ovarian, uterine, and even cutaneous nerves, has, in each instance, been proved by more or less precise experimentation. The physiological sympathies of the heart being thus extended, it is not to be wondered that functional disturbances of almost every organ may entail derangement of cardiac action and feeling. Of feeling, for there seems little doubt that the heart, though unendowed with animal sensibility in health, may acquire it in disease and under the influence of special impressions.

1164. Hysterical and anæmic palpitation, accompanied with intercostal neuralgia, nay, even organic palpitation in a person with that form of neuralgia,* may readily be mistaken for true angina; indeed, an attempt has been made by some French writers to show that the latter disease is nothing more than a "brachio-thoracic neuralgia," the heart-symptoms being purely accidental. In true angina, the points of tenderness in the course of an intercostal nerve are wanting; besides, the severity of the suffering in the cardiac region is infinitely greater than in intercostal neuralgia.

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* Roberts, U. C. H., Females, vol. v., p. 217; Hawkesford, id., vol. v. p. 1.

1165. Although common-sense and experience point to the necessity of learning the cause of pseudo-angina, in order to cure the effect, still, in instances where we either fail to discover that cause, or find it to be itself non-removable, much good may be done to the cardiac symptoms by direct treatment of these on the principles applicable in cases of true angina.

PASSIVE AND MECHANICAL CONGESTIONS.

1166. Passive and mechanical congestions of the heart's tissue and membranes, however interesting to the morbid anatomist, are without clinical importance in the present state of knowledge; there are, in fact, no known means of diagnosticating these states.

1167. But congestion of (or, rather, accumulation of blood in) the cavities of the heart is a state at once productive of serious symptoms, and clinically demonstrable. Such accumulation may occur in any, or in all, of the cavities, in cases of endocarditis, as a consequence of fibrinous particles interfering with the free play of the valves,—in cases of polypoid concretions, of whatever origin,—and in cases of rupture of valves. Accumulation in the right cavities, especially, will ensue in prolonged fits of palpitation, with highly-disturbed rhythm,—in cases of tricuspid regurgitation,—during fits of dyspnœa, in highly-marked emphysema of the lung, especially if this has already led to organic dilatation of the right cavities, —and, probably, to a greater or less extent, in all cases of suddenly obstructed circulation through the lungs.

1168. The symptoms associated with such loading of the right heart are dyspnœa even to orthopnœa, dry cough, venous congestion of the face and upper surfaces generally, unattended, unless there be prior anasarca of the lower extremities, with œdema; oppression, anxiety, and sometimes pseudo-anginal feelings.

1169. The heart's impulse, laboured and struggling, irregular in force and rhythm, is seen and felt more extensively than natural, especially to the right of the sternum, and at the epigastrium. The area of dulness exceeds that of health, especially about the right costal cartilages.

1170. Venesection, cupping over the præcordial region, sharp, rapid purgation, counter-irritants to the lower limbs, or the application of Junod's apparatus, are the remedies theoretically indicated for the relief of this state; practically, too, they prove efficient. But the condition, on which the obstruction of the cavities depends, remains of course in the back-ground, unmodified, or scarcely modified, by them. Ulterior measures must be taken for the removal of that condition, if possible.

ACUTE PERICARDITIS.

1171. The anatomical stages of completely evolved acute pericarditis are five: those of dryness with vascularity; of plastic exudation; of liquid effusion; of absorption; and of adhesion.

1172. The dry stage is marked by florid, arborescent, and capilliform injection, sometimes by prominent tufts of capillary vessels,* especially on the cardiac portion of the sac; the membrane itself is dry and parchment-like. This stage is very rarely seen except where persons dying of other diseases are seized with pericarditis, just before dissolution.

1173. In the exudation-stage plastic lymph of various degrees of firmness accumulates on both surfaces; on one surface, namely the cardiac, I have never seen lymph limited to the parietal division; on the posterior aspect only of both cardiac and parietal portions; on the anterior surface of the right ventricle only; or specially about the roots of the great vessels. The lymph is laid down stratiformly, thick, thin, or in lace-like fashion, resembling the intra-cranial osteophyte of pregnant women; or in hillocks; or in regular layers separated by indentations; or locularly; or in loose thread-like manner. Three-quarters of an inch is the extreme thickness of lymph I have witnessed, and this only about the great vessels. Essentially of straw colour in itself, it is reddened by vascularity and imbibition of blood.

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* F. Parker, U. C. H., Males, vol. iv., p. 177. Dec. 1848.

1174. The liquid effusion of the next stage may be sero-flocculent; sero-fibrinous and spontaneously coagulable after removal from the sac; hæmorrhagic; sero-purulent,—pus in sufficient quantity to be recognised with the naked eye being rare; or purulent. The quantity of fluid ranges between two or three and sixty ounces,* the greatest quantity I have ever witnessed. The liquid macerates, softens, rarifies and distends the pericardial membrane. Sloughing is very, pneumo-pericardium from decomposition of the fluid infinitely, rare.

1175. In the absorption-stage serous fluid is removed with ease and rapidity,—hæmorrhagic effusion with less facility, and purulent with still greater difficulty. Exudation-matter of variable plasticity, to form the material of adhesions, agglutination, and the nidus of various Precipitates and Pseudo-Tissues, remains; or in some cases even exudation-matter is freely removed by absorption.

1176. In the next stage the two pericardial surfaces may become universally agglutinated together; or locally adherent, either closely by patches or loosely by bands; adhesion of the great vessels is common. Or exudation-matter solidified may exist without adhesion occurring: this is particularly observed about the great vessels, where such matter may long remain the source of physical signs above the heart's base. Locular or meshlike adhesions, containing fluid, absorbable and renewable, sometimes form.[*]

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* Bartlett, U. C. H., Males, vol. iv., p. 292.

1177. The ulterior changes in the sac belong, clinically, to the chronic disease.

1178. The secondary lesions of pericarditis are endocarditis, carditis, local pleurisy, pneumonia and bronchitis: the various cerebro-spinal symptoms sometimes observed are not assignable to any textural change in the nervous centres.

1179. Pericarditis is rarely an isolated disease, and rarely springs up in a person of sound constitution: the conditions under which it originates may be set down as follows:—

1.	Idiopathic.	
2.	Traumatic	Wounds of pericardium from without, or through œsophagus;[†] blows and contusions of præcordial region.
3.	Perforative	From liver-abscess through diaphragm.[‡]
4. Attendant on or consecutive to—	General diseases	Typhus, typhoid fevers, variola, scarlatina, pyohæmia.
	Diathetic diseases	Rheumatism, Bright's disease, gout, scurvy, purpura, cancerous and tuberculous diatheses.
	Diseases of blood-vessels	Phlebitis, endosteal and other, after amputations and various operations.
	Peculiar states. the blood	Cyanosis.
	Sudden removal of established diseases.	Psoriasis, chronic eczema, and lepra.
	Diseases of pericardium	Cancer, tubercle.
	Adjacent diseases	Pneumonia, pleurisy, inflamed mediastinum, abscess with probably diseased rib.[§]

1180. But though I have seen, or read authentic accounts of, pericarditis arising under all these conditions, practically speaking the disease is scarcely met with in London but as a local development of the rheumatic diathesis and that of Bright's disease. In rheumatic fever the joints commonly suffer before the pericardium; or the pericarditis may lead the way, the joints following; or both pericardium and joints may suffer simultaneously; or there may be no arthritic affections, pericarditis and acid sweats existing alone.* The late Dr. Taylor proved that Bright's disease in its advanced stages acts almost as frequently as a cause of pericarditis, as acute rheumatism. Scurvy in some parts of northern Europe appears to be a very frequent cause of pericarditis. The tuberculous and cancerous diathesis may very rarely generate the disease. Alleged idiopathic pericarditis becomes rarer every year, in proportion as the evolution of diathetic diseases grows more fully understood; I have never seen a positive case of the kind.

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[*] M. Corvisart's recent observations on the closed sacs, probably glandular, at the base of the pericardium, originally described by Lower, and M. Remak's detection of ganglia on the cardiac surface, are possibly destined to enlarge our notions of the anatomy and pathology of pericarditis.

[†] U. C. Museum, No. 3859.

[‡] Graves, Clinical Medicine.

[§] Newman, seen with Mr. Quain, U. C. H. Jan. 1854.

It is not uncommon to find the various co-existences, above enumerated, spoken of as complications of pericarditis,—a lax use, it will be conceded, of the term.

1181. *Physical signs.*—During the *dry stage*, the extent of visible impulse is greater than natural; the impulse, as felt, is too forcible, of beating rather than heaving character, and successive impulses are of unequal strength: the action is suggestive of excitement. The areas of dulness, both superficial and deep, are unchanged. Grazing friction-sound may occasionally be caught; and even sharp, though feeble, scraping-sound, if there be a prominent tuft of dilated capillary vessels.† The physician should, at this period, while it is yet unchanged by the disease, accurately ascertain the point of the apex-beat, in order to substantiate its subsequent displacements.

1182. In the *plastic exudation-stage*, it is said by some observers, that incipient bulging of the præcordial region may be noticed; muscular paralysis from adjacent inflammation, *plus* the protrusive action of the heart, sufficing for its production. I have never succeeded in discovering this; nor does the theory seem at all sound: admitting that the inflammation of a serous, in close contact with a muscular membrane, paralyses the latter, as in the alleged instance of pleuritis, it does not follow that pericarditis will have any such effect, seeing that the pericardium is not in contact with the side. The inspection-signs are essentially the same as in the dry stage; but elevation of the apex may be effected by the contraction of lymph solely, if this stage be protracted [347]: the wings of the diaphragm may be somewhat raised too. The hand sometimes detects pericardial thrill.* If the plastic exudation be very thick, it is conceivable that the area of superficial dulness shall be extended; but this is a point too delicate to be trusted to. The essential sign of this stage is pericardial friction-sound, of which the properties have already been described [457, 474]. The condition of the heart's sounds varies; they may be unchanged, or even louder than in health; or, on the contrary, masked somewhat by the loudness of the friction-sound, or even positively enfeebled, especially the first, in all probability by the interference of thick layers of lymph with the full play of the ventricles. Valvular murmurs are of excessive frequency as dependencies on co-existent endocarditis,—especially the aortic constrictive and mitral regurgitant varieties. But may valvular murmur come directly of exudation on the pericardial surface? It is conceivable that the aorta and pulmonary artery may be so pressed on by lymph, that murmur shall be engendered with the systole as the blood passes through the slightly constricted part; but I do not know this from experience: in regard of prognosis, the question is obviously one of importance,—a murmur, thus generated, must be of less serious import than one of endocarditic origin. The respiration-sounds continue unchanged over the heart.

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* Perry, U. C. H., Females, vol. i., p. 82.

† Parker, U. C. H., Males, vol. iv., p. 177.

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* I have never observed thrill produced by cardiac action on pleuritic ymph, the pericardium being positively healthy: Dr. Stokes, however, appears o have done so (Op. cit., p. 29).

1183. The perfection of the signs of the *stage of liquid effusion* varies directly as the amount of fluid. By inspection may be discovered arching of the præcordial region, widening and even bulging of its intercostal spaces, with elevation of the left edge of the sternum, sometimes traceably increasing from day to day; œdema of the præcordial integuments, especially if the effusion have existed for any time; undulating impulse; and displacement of the apex-beat upwards as far as the fourth interspace, and slightly outwards. By application of the hand we find that in cases where the apex lies behind a thick stratum of fluid, the impulse lags slightly behind the systolic sound; the impulse feels weak, unequal, fluttering, or may be imperceptible; if pericardial thrill had existed, it is now gone; the line of vocal fremitus at the right side of the heart is carried unnaturally to the right,—a valuable sign in some cases; the state of respiration-expansion over the heart varies,—if the quantity of fluid be moderate, costal expansion is well marked, diaphragmatic movement being impeded by the fluid,—if very great, that expansion is impaired. The interval between the left nipple and the middle line may be increased; but this is not always the case, even where bulging is well marked. Percussion discloses what is, all things considered, the least fallacious sign of effusion, namely, præcordial dulness of the peculiar pyramidal form and other properties already described [381]. The area of this dulness may be changed sidewards, most readily to the right, by moving the patient successively from one side to the other.

1184. By auscultation, the irregularity of the impulse in regard of force, and, if this be affected, of rhythm, is better perceived than by other means. The friction-sound of the past stage may be either completely gone; or heard in some spots about the great vessels; or pretty generally retained in the præcordial region,—but this is very rare even with eight ounces of fluid, and it is scarcely possible with more than ten. On the other hand, *no conceivable amount of fluid will of necessity totally annul friction-sound.* I base this statement on a case in which I and others distinctly heard friction-sound "at mid-sternum on the level of the third rib," and yet (death occurring only twenty-nine hours later) sixty ounces of fluid were found in the pericardial sac, which reached about a thumb's breadth *above* the clavicle.* The possibility of systolic basic murmur being produced by pressure of fluid on the great vessels has already been referred to [437]. The heart's sounds feeble, distant, and as it were muffled, at the lower part of the cardiac region, become louder as the stethoscope is carried upwards, and at the top of the sternum the second sound is full and clear, and the first very decidedly more marked than directly over the ventricles.

1185. The anterior edges of the lungs are pushed aside by the accumulating fluid; and the left lung may be pushed upwards, it would appear from a case observed by Dr. Stokes, so as to form a tumor above the clavicle, where there is sudden copious effusion both in the pericardium and left pleura. The central tendon of the diaphragm undergoes depression, and may be rendered convex inferiorly; the liver *may* be pushed downwards and to the right, but an enormous amount of fluid will not necessarily displace it.

1186. The respiration-sounds in the centre of the cardiac region are feeble and distant: in some very rare instances the voice resounds with an ægophonic twang at the edge of the effusion: this is especially likely to occur, if the adjacent border of the lung be indurated.

1187. In the *stage of absorption* undulatory impulse disappears; the point of the apex-beat almost invariably falls; the bulging of the cardiac region gives way. By the hand we ascertain that the impulse has recovered its breadth and fulness; friction-fremitus, too, may return. The dulness of effusion gradually diminishes from above downwards, and draws in laterally also, but not till it has undergone a very distinct fall superiorly. Redux friction-sound is caught by the ear, commencing about the roots of the vessels, and varying in extent with the rapidity of absorption. The churning or continuous rumbling variety [459] is the rarest condition of sound discovered. The heart's sounds recover their fulness (if endocarditis have not prominently existed), and also their natural nearness to the surface; the respiration returns slowly, and may never, especially if agglutination of the pleural surfaces occur in front of the pericardium, recover its natural intensity.

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* Bartlett, U. C. H., Males, vol. iv., p. 292.

1188. The occurrence of the last stage, that of *adhesion* of the pericardial surfaces, is announced by disappearance of ordinary friction-sound, and *à fortiori* of friction-fremitus, if this have existed; the former may be clearly heard in some points, where adhesion is as yet unestablished. Besides, the clicking variety [460] may long continue audible at the base. The percussion-dulness continues to decrease, or at all events does not increase, in area. The action of the heart may be tremulous, unsteady, or jogging.

1189. The moment adhesion is accomplished, the evolution of the acute disease has reached its final term.

1190. *Symptoms.*—The chief *local* symptom of pericarditis is pain, occupying the cardiac region only, mainly seated in the epigastrium, or extending to the left shoulder and elbow,—slight in amount or of agonizing severity, lancinating, tearing, burning, or consisting of a mere sense of soreness; and increased by movement, and deep inspiration. Pain, however, may be absent, and in the majority of cases is either absent or of slight severity. The varying amount of suffering in different cases is with difficulty accounted for; coexistent phrenic pleurisy seems to explain the acute agony of some patients, but pleurisy *may* be present and the pain moderate, or *vice versâ*. Rheumatism of the diaphragm, phrenic or intercostal neuralgia or neuritis, are all conceivable, but non-proven, causes of excess of suffering. The præcordial interspaces are generally tender; pressure in the epigastrium causes great distress. Palpitation is not usually a prominent symptom, and may be totally deficient, especially as a condition of which the patient is painfully conscious.

1191. (*a*) The decumbency is least commonly on the left side, most commonly on the back; the head is generally kept rather high. Orthopnœa, a most inconstant symptom, if present, is not any proof, as has been taught, that liquid effusion has taken place; it may be absent during the effusion-period, and first appear after absorption; again, it may occur paroxysmally and irregularly. Far from orthopnœa being a necessary result of effusion, the patient, where this is most copious, may lie by choice flat on the back, with scarcely any pillow: this was the habitual posture, for days before death, in the case just referred to, where sixty fluid ounces had accumulated in the sac. In such cases, the least elevation of the head produces a tendency to syncope,—and the patient instinctively dreads movement of any kind. The facial expression is generally anxious; the features drawn: in fatal cases, *risus sardonicus* sometimes occurs towards the close. The sleep is fitful and disturbed; and jactitation of the arms—the trunk being kept quiet—not unfrequent in serious cases. Apprehension of death may be a prominent symptom. (*b*) Rigors may announce the invasion of the disease, but they are not commonly severe nor repeated; the skin, subsequently hot as the usual state, may be paroxysmally cold; if perspiration occur, it is not specially acid, unless rheumatism be present,—and it *may* be alkaline, even with this combination. Sudamina sometimes form, and their contents may be alkaline, while the surrounding perspiration is acid,—or the reaction of both may be the same: none of these conditions are peculiar to the disease. Œdema sometimes occurs, particularly if the case be protracted, about the ankles; and may also appear in the integuments of the cardiac region. The integuments of the head, face, and neck, may become markedly livid with swelling of the subcutaneous veins. (*c*) The joints are not affected as a consequence of pericarditis,—they are frequently rheumatic, of course, as the majority of cases of pericarditis are of rheumatic origin. The limbs generally are the seat of febrile pains. (*d*) There is nothing special in the state of the tongue. Spasmodic dysphagia sometimes occurs. Tenderness of the epigastrium, with nausea and vomiting of food, or of bile even (the stomach being perfectly sound,*) sometimes constitute very prominent symptoms. The liver grows engorged; I have never seen jaundice. (*e*) Dry, irritable, abrupt, jerking, spasmodic cough, with variable dyspnœa, and coolness of the expired air towards the close, are the chief pulmonary symptoms; that exaggerated respiration results from pericarditis *per se*, I more than doubt. The voice is sometimes much weakened,—an effort required to produce feeble tones: † this symptom seems mainly connected with copiousness of effusion. (*f*) The pulse is commonly frequent out of proportion with respiration, unless there be some pulmonary complication. I have, however, known in rheumatic pericarditis, with chorea, a ratio of 136 : 64 or 2·12 : 1, the lungs being free from inflammation.‡ At first full and hard, and sometimes less frequent than natural, afterwards weak and feeble, the pulse grows irregular, both in force and rhythm, in about one-third of cases,—or such irregularity may exist from the first, and before fluid is present to account physically for the circumstance,—or irregular and uncountable from frequency one day, it may be infrequent and perfectly regular on a subsequent one, the disease meanwhile advancing.§ The frequency of the pulse is subject to more sudden variations, from the influence of emotional excitement and effort, than in any other disease perhaps: thus I have known a very gentle movement of the trunk raise the pulse from 80 or 90 to 130 or 140. The blood is hyperinotic. (*h*) The urine is febrile, or possesses the characters of the diathetic disease, with which the pericarditis is associated. (*k*) Cephalalgia, though present, rarely excites spontaneous complaint, and all serious head symptoms may be totally absent from first to last. But, on the other hand, delirium, apoplectiform stupor with imperfect hemiplegia and a quasi-maniacal state* or actual mania of violent character, sometimes occur. So, too, serious symptoms of spinal character occasionally appear: as cramps, slight convulsions, epileptiform, hysteriform, trismal and tetanic seizures, and chorea in all degrees of severity.

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* G. Perry, U. C. H., Females, vol. i., p. 82. 1846.

† Bartlett, U. C. H., Males, loc. cit.

‡ Leason, U. C. H., Females, ætat. 9, vol. viii., p. 69.

§ Bartlett, U. C. H., Males, vol. iv., pp. 295-6. Rhythm may be regular, while force is irregular.

1192. These cerebro-spinal symptoms are sometimes in all likelihood produced rather by the altered state of blood coexistent with the pericarditis than by the local conditions of this inflammation in itself: in the pericarditis of Bright's disease uræmia is probably their immediate cause. In the rheumatic variety these symptoms may be of reflex mechanism from irritation of the phrenic nerves,—a view, though deficient in proof, certainly more tenable, than that ascribing them to cerebro-spinal meningitis. When this latter condition actually exists, no one of the symptoms just enumerated may occur.† Since I first noticed dysphagia as a symptom of pericarditis in the former edition of this work, I have seen two additional striking examples of it: ‡ in one of the patients, an adult, wildness of manner, tremors, paroxysmal orthopnœa and faintness and subsultus tendinum coexisted; in the other, a child, violent bilateral choreal movements. Both cases confirm the opinion formerly expressed that the symptom is of spasmodic character.

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* Campion, U. C. H., Females, loc. cit.

† Gash, U. C. H., Males, vol. x., pp. 178—182.

‡ Campion, U. C. H., Females, vol. vii., p. 260; and Miss B——, seen with Dr. Neil Arnott.

1193. *Course.*—Pericarditis may run a uniform course, with periods of increase, status and decline; or undergo suspension as regards subjective and many objective symptoms. Relapse is not common; whereas, as far as I have seen, recurrence is more common than with any other inflammation of serious character.

Pericarditis may run a latent, or an obvious course,—the former more commonly in the diathesis of Bright's disease, than under other circumstances. Latency of course may occur under all anatomical conditions of the disease,—and seems plainly connected with the special nervous susceptibility of the individual, rather than with the amount of local mischief.

1194. *Terminations.*—When pericarditis terminates clinically by recovery, there is reason to believe, as acutely shown by the late Dr. Taylor, that, anatomically, actual resolution sometimes takes place; more usually absorption of fluid, with plastic adhesions, ensues,—or, without such adhesion, where one surface only has thrown out lymph.

Or the acute passes into the chronic disease, with adhesion and agglutination,—and more or less constant tendency to irritative congestion.

Or death occurs. Commonly syncopal, death may be *gradually* effected either by the mechanical interference of the fluid with the heart's action, or perhaps by that of effused lymph. It seems wonderful what an amount of fluid can be borne with impunity by the heart. Fifty to sixty ounces do not necessarily stop its contractions. But this obviously depends on the slowness of its accumulation and extensibility of the pericardium: a few ounces of blood suddenly poured out will, we know, mechanically destroy life in an instant. Contraction of effused lymph may so twist and distort the heart, that mechanical suspension of projection of the blood, through the orifices, becomes well conceivable.* Or death may *suddenly* take place from raising the head, or otherwise moving abruptly,—possibly from a sort of cardiac paralysis. Lastly, the fatal issue may be traceable to cerebro-spinal disturbance.

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* Campion, U. C. H., Females, vol. vi., pp. 40, 41.

1195. *Duration.*—In fatal cases death may occur within thirty-six hours: Andral records a case fatal in twenty-seven from the first supervention of pain; Mirabeau was cut off by pericaditis so rapidly, that the reports of his having been poisoned readily gained credence. In instances of recovery disappearance of subjective, and of most of the objective, symptoms takes place in from twelve to twenty days; but physical signs may hold on for weeks, nay for months, especially slight dulness, and clicking friction-sound, above the heart's base.

1196. *Diagnosis.*—The diagnosis of pericarditis can only be made with security by the physical signs. For, first, so absolutely *latent* from first to last may the disease prove, that I have known patients with several ounces of fluid and exudation-matter in the pericardium, grow irritated, when inquiries were made about symptoms connected with the heart; secondly, there may be a total want of harmony between the violence of the symptoms of which the patient is actually conscious and the amount of disease; or, thirdly, the symptoms of other affections may be simulated.

1197. Before the occurrence of friction-sound there is no certainty in the diagnosis. But if there coexist with sudden excitement of the heart, and absence of endocardial murmur, præcordial distress and tenderness under pressure, while the pulse is frequent (I have not been able to confirm Dr. Graves's observation as to its infrequency), and the respiration non-accelerated, the probabilities pronounce for pericarditis.

1198. The essential signs of pericarditis are friction-sound, special præcordial dulness, and twisting upwards of the heart's apex. Friction-sound, when thoroughly developed with the characters assigned it elsewhere [458-470], is next to pathognomonic: the possible fallacy from pleural friction of cardiac rhythm [473] must not, however, be forgotten. Mediastinal pseudo-rhonchus, which may coexist with pericardial friction too (case of Ramo Samee), and the squashy rhonchoid sound, sometimes produced by pressure of the stethoscope over the heart, when the integuments are œdematous, are other possible sources of difficulty. The rules already laid down [474] will commonly distinguish pericardial friction from endocardial murmur: since that paragraph was printed, I have, however, found that even mitral murmur may be intensified by pressure with the stethoscope.* On the other hand, pericardial friction may really be heard, and mistaken for other things. The clicking variety differs, however, from valvular clicks in its non-synchronism with either heart-sound, and in its non-transmission along the aorta. Again, exudation may be present, and yet no friction-sound evolved,—either because the posterior aspect of the heart only is affected,—or because one lamina of the membrane only is coated with lymph,—or because recent agglutination has occurred,—or because old agglutination prevents attrition,—or because serum or pus has been copiously poured out.

As concerns percussion-results, if the peculiar pyramidal dulness [381] be developed under the eye of the physician, and have succeeded to friction-sound, there is no possible source of fallacy. But if it have not been preceded by friction, the dulness may be from hydropericardium simply; and if the pyramidal form have not been produced under observation, there are certain sources of fallacy of a serious character already mentioned [381]. Thus a weak fatty heart, with quasi-undulatory impulse and feeble sounds, intermittent pulse, and febrile action, may exist, where the form of the heart's dulness is rendered triangular by the chance presence of old exudation-matter about the great vessels, a tumour, a small solid quiescent aneurismal sac,—or even of an excess of natural mediastinal fat. How is the distinction of the cases to be established? Probably, in the whole range of thoracic diagnosis, there does not exist a more difficult problem. Thus, an adult labouring obviously under acute disease, unable to give any trustworthy account of himself, had all the physical heart-signs mentioned, and in addition orthopnœa and jactitation, while, on his admission to the hospital, no ordinary signs of pneumonia or other thoracic inflammation existed to account for the acute aspect of the disease. I was strongly disposed to regard this as a case of latent pericarditis with effusion, accompanying valvular disease, of which the signs were obvious; but the impossibility of discovering a shadow of friction-sound, though the posture was varied, the non-elevation of the heart's apex, and the fact that above the third rib the dulness was not so absolute as below it, led me to reject the idea. On *post-mortem* examination (the signs of pneumonia had meanwhile made their appearance,—on admission, indeed, the pulse-respiration ratio was 2·7 : 1, and in twenty-four hours had become 2 : 1), the pericardial sac was found free from fluid; but above the base of the heart lay a lump of fat,* the simple source of all the difficulty. And it is quite conceivable this difficulty might be increased;—for a patch of old exudation-matter near the apex might give friction-sound, while all the other conditions were essentially the same as in the case just referred to.†

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* Beere, U. C. H., Females, vol. x., p. 99; Craddock, U. C. H., Males, vol. x., p. 83.

1199. Mediastinal tumor of some size, if it encroach on the cardiac region, may simulate pericardial effusion: the history of the case; the presence of centripetal pressure-signs, which are never caused by fluid in the pericardium; and the outline of the dulness, which can scarcely by an unlucky chance imitate precisely that of pericardial dulness, (a tumor grows in spite of gravity, fluid obeys this,) will distinguish the former from the latter.

1200. Here is another puzzling combination: a female may have fever, dry red tongue, extreme epigastric, and no præcordial, tenderness, bilious vomiting and diarrhœa, and perfectly regular pulse,—present not a single symptom connected with the heart, and be free from Bright's disease or acute affection of the joints. How is this state of things, really dependent on pericarditis with effusion, to be distinguished from acute gastritis? The physical signs say nothing: friction-sound is gone, when the patient is first seen, and a huge stratum of subcutaneous fat and a massive mamma may deprive us of the evidence derivable from percussion.*

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* Beckett, U. C. H., Males, vol. v., p. 229.

† J. Morris, U. C. H., Males, vol. vii., pp. 154—162.

1201. In the diagnosis of a difficult case, the functional and general symptoms should not be forgotten,—but clinical experience compels us to admit that they are sometimes utterly fallacious aids. Hope wrote the singular proposition, that "the variability of the symptoms is calculated rather to enlighten than perplex the practitioner,"—his belief being that the symptoms of lymph-deposit were slight, of effusion serious. The value of this dogma appears from a single case, the fourth in Andral's Collection, where death occurred from plastic pericarditis in twenty-seven hours, without a drop of fluid having formed in the sac; while *per contra* the disease may be latent with extensive hydro-pericarditis.

1202. *Mortality and Prognosis.*—There are no existing documents from which the absolute mortality, or the ratio of deaths to seizures, may be safely calculated on a large scale.

1203. The *immediate* prognosis is in the majority of cases not unfavourable. M. Louis long since estimated the deaths at one in six of those attacked.† But the result is greatly under the influence of the existing diathesis. I do not remember to have seen rheumatic pericarditis fatal, in a previously healthy person, except in some seven or eight instances; whereas a large proportion of cases of pericarditis, arising in Bright's disease, end fatally. Dr. Taylor found in seventeen cases of recovery the mean age = 19·7 years; in twenty-one cases of death = 35·5 years: but renal disease would be more likely to exist in the series of more advanced age than in the other,—age alone cannot, therefore, be charged with the difference in mortality. The whole class of reflex phenomena are of excessively bad augury: when highly marked choreal symptoms have occurred in childhood, I have never known recovery ensue. Dysphagia at any age is of very fatal significance.

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* All these facts were illustrated by the case of E. Perry, U. C. H., Females, vol. i., p. 82.

† Mémoires Anat. Path., p. 291. 1826.

1204. In regard of *remote* prognosis: if mere adhesions or even agglutination exist, the health does not necessarily suffer; if hypertrophy, simple or dilated, or atrophy, supervene, symptoms of course arise. M. Beau found in forty-eight cases of agglutinated pericardium forty examples of dilated hypertrophy, but the relative dates of the two conditions cannot always be established from his facts. I have seen no reason to accept the doctrine that old pericarditis in itself kills: coexistent endocarditis with its valvular sequelæ is the real destroyer in chronic fatal cases.

1205. *Treatment.*—The treatment of pericarditis will in some wise be modified by the diathetic state it accompanies. If it appears in the course of rheumatism, either after, along with, or before the articular affections, possibly the intensity of the inflammation might be lessened by artificially irritating the joints. To grant this, obviously does not require one to have any faith in the exploded doctrine of metastasis; but the *practice* has disappeared, perhaps unmeritedly, since the *theory* has been banished from the schools.

1206. Bloodletting takes rank among the foremost remedies in pericarditis; but in judging of its utility in any particular case, the causes of the inflammation must be borne in mind: rheumatic pericarditis in the great majority of cases terminates favourably, no matter what the precise mode of scientific treatment adopted; pericarditis of renal origin almost as invariably proves fatal. Bleeding from the arm is attended with a certain amount of risk of syncope; it does not prevent other inflammations coming on, be it ever so free; very copious depletion in certain constitutions excites the heart greatly, and in some respects makes matters worse; and the most severe rheumatic pericarditis may go calmly on to recovery, though moderate cupping over the præcordial region has been substituted for venesection. Whether bleeding at the outset may ever arrest the disease at once, I do not believe to be scientifically ascertained: it may lessen pain and distress without a jot abating the activity of the exudation-process. That the curative importance of copious bloodletting has been exaggerated, while its special dangers were ignored, by Dr. Hope and others, no reasonable doubt can at the present day be entertained. In the fulness of his enthusiasm, Dr. Hope wrote, "the loss of a few hours at first may be irretrievable, and hence hesitation and indecision may seal the fate of the patient. Venesection to the verge of syncope, followed by twenty-five to forty leeches," he holds to be essential in severe recent cases; and "unless the pain be completely subdued by these measures, the leeching, and in some cases the general bleeding also, may be *repeated two, three, or more times.*" What then is to be said of a case, which I have elsewhere published, where violent acute rheumatism coexisted with pericarditis, endocarditis, double pneumonia and pleuritic effusion, and probably aortitis, and yet, twenty-two ounces only of blood having been taken by cupping from the cardiac region, convalescence was established by the twenty-first day?* I never draw blood largely from the arm in rheumatic pericarditis; and, as I formerly said, "the reasons why I abstain from the practice are not that I fear people profusely bled shall 'forthwith go raving mad,' (as they have done, nevertheless, but in rare instances); nor that I fear endocarditis shall forthwith be generated,—an effect which the practice has never been proved to entail. My reasons are, first, that none of the advocates of such venesection have ever shown that the mortality, duration, or suffering, of the disease, are less in a series of cases thus treated, than in a series of cases treated by gentler means—by colchicum, alkalies, and mercury, for instance. And secondly, that while, by prodigal bleeding, a loss of vital fluid is inflicted, which it may take the nutritive system months, or even years in individuals of certain constitutions, to repair, all that loss may be saved without demonstrated risk of any kind."

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* Clinical Lectures, case of Craddock, Lancet, Feb. 1849.

On the other hand, that moderate venesection shortens the duration of pericarditis, and does so more effectually the earlier it is performed, has been clearly shown by Dr. Taylor in his logical papers on the treatment of the disease. The quantity of blood to be drawn must be regulated by the severity of the symptoms: from an adult of medium strength (it is to be remembered that loss of blood is worse borne in renal than in rheumatic pericarditis) some twelve to sixteen ounces may be taken from a vein in the arm, the head being kept *low*, especially if there be much fluid in the sac. This depletion may be followed, if well supported, by the abstraction of some six or eight ounces more by cupping or leeching over the heart. In cases of slight severity, local bleeding may be most confidently trusted to alone.

I have known moderate bloodletting at once improve the tone of the first sound, probably by exciting absorption of fluid in the sac, and facilitating cardiac action.

1207. The accurate evidence before the profession concerning the influence of mercury, though it leads us to question the extreme power of the mineral, nevertheless shows that its administration is not to be neglected. It stands obviously second to bloodletting,—and appears to carry out, as it were, the good effects produced by this. Salivation is with difficulty induced, as is well known; although no positive proof exists that the utility of the mineral is measurable in this disease by the rapidity of ptyalism, still, in obedience to general conviction, it is well to ensure this result as speedily as possible; and this may best be done by the plan recommended in a previous paragraph [691]. Ptyalism being effected, the disease is not necessarily arrested; Dr. Taylor refers to three cases in which increase of signs and symptoms distinctly followed.*

1208. On the principle of regarding the disease through its diathesis, colchicum and alkalies are advisable, where the pericarditis is rheumatic; I do not think colchicum should ever be omitted in a case of the kind, and it may be given in the form of draught along with the mercurial pill. Opium becomes a necessary remedy in full doses, should agitation and disquietude be at all marked; morphia may be used endermically, if it has been found advisable to apply a blister to the præcordial region. The application of a blister there is, however, objectionable from its interfering with the examination of the cardiac region, and hence preventing a precise knowledge of the state of the disease; blisters should rather be applied behind, than actually on, the præcordial region. Digitalis, aconite, and hydrocyanic acid are dangerous agents, from the chance of their increasing tendency to syncope. Purgatives, diuretics, and diaphoretics are advisable as aids in the treatment.

1209. Sinapisms frequently repeated are of great service in relieving pain and distress, and are not open to the objection just mentioned in the case of blisters. Ioduretted frictions, coupled with mercury in very small proportion, seem to promote absorption of exudation-matter, especially when the more acute state has passed by.

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* Dr. Taylor's evidence, indeed, so far as it goes, rather bears against mercury: he refers to the frequent occurrence of acute inflammations during salivation for the cure of others. I have known pericarditis supervene in a woman while under treatment for ptyalism, so severe, that for some hours after her admission into hospital for the cure of that ptyalism (Spratt, U. C. H., Females, vol. iv., p. 471) life was in danger from semi-asphyxia, and tracheotomy on the point of being performed; but such cases must be esteemed singular exceptions. Mercury is supposed to act in inflammations by diminishing the quantity of fibrine, yet M. Andral found hyperinosis in four cases of mercurial stomatitis (Hématologie, p. 90).

1210. The hot-air or vapour bath, in the pericarditis either of rheumatism or Bright's disease, is a valuable adjuvant,—both have the advantage over the warm bath, that they may be taken with the head moderately low.

1211. The regimen must be strictly antiphlogistic; and the patient strictly cautioned, in the advanced stages, against abrupt movements and elevation of the head.

1212. No special treatment is required for the various inflammations sometimes occurring secondarily. But the whole class of reflex symptoms call for the suspension of depletory measures and of mercury; purgatives, antispasmodic and sedative remedies must at once be had recourse to; and an attempt made in rheumatic cases to irritate the joints by sinapisms. If dysphagia arise, the neck should be blistered.

CHRONIC PERICARDITIS.

1213. Under the head, chronic pericarditis, may be included four states clinically very different: namely (*a*), that in which adhesions or agglutinations of the pericardium having formed, a tendency to active congestion in the pericardium itself, and in the substance of those adhesions, is more or less constantly present; (*b*), that in which cardiac hypertrophy is conjoined with such adhesions; (*c*), that in which cardiac atrophy coexists with such adhesions; (*d*), and that in which liquid effusion remains in the sac without apparent inclination to increase or to disappear.

1214. (*a*) In the first form, that of adhesion, there may be, in the absence of irritation, a complete nullity of symptoms,—neither in cardiac action subjectively considered, nor in the state of feeling about the heart generally, is there anything to arrest attention. If there be congestive tendency, palpitation is easily excited, the breath short, and uneasiness of various kinds readily excited.

1215. We have seen what the physical signs of adhesions are at the time of formation; they are easily established. But in a case, seen for the first time, after adhesions have been some time formed, their positive diagnosis is among the most difficult clinical problems existing. The heart itself being either quite healthy, or not seriously changed in bulk or structure, the signs will vary, first, with the closeness of the pericardial adhesions themselves,—and secondly, with the absence or presence of pleuritic adhesions in front of the heart. So essential is the latter point, though ignored by writers generally on the subject, that it forms the ground of a distinction of the physical signs into two classes, as follows:—

Pericardial Pleuritic Adhesions

Absent.	*Present.*
Apex-beat in natural site, or retained above, in the spot it was raised to during the acute stages.	The same.
Apex-beat not notably lowered by inspiration.	This peculiarity still more marked.
Depression of præcordial region, about fifth to seventh left cartilages, or superiorly at the base of the heart.[*]	This more marked.
No dimpling of surface with cardiac action.	Epigastrium at left costal angle [12] may dimple inwards with the ventricular systole.
No undulating movement of heart.	Undulating movement more or less well marked.
Respiration-movements slightly less active over heart than natural.	Respiration-movements almost null over heart.
Percussion-dulness about large vessels above third left cartilage from induration-matter.	This still more marked from pleural induration-matter, and condensation of neighbouring lung-substance.
Pericardial clicking sounds about roots of vessels, or common friction-sound from loose adhesions.[†]	The same.
Complete agglutination may exist without jogging, trembling motion of the heart.	Jogging trembling motion of the heart: (still, even here, this is rare, if the heart's bulk be natural).

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[*] Case of Craddock, Clin. Lectures, Lancet, loc. cit.

[†] Kennedy, U. C. H., Males, vol. i., p. 67 [470].

It has been said that feebleness, even to extinction of the second sound, is a sign of pericardial adhesion: I doubt whether the two things, when associated, have ever any direct connection; and I know that complete agglutination may co-exist with a perfect second sound.

If new effusion occur in a formerly inflamed pericardium, the continuance of respiration in the præcordial region is a sign of adhesion, pericardial and adjacent pleuritic combined.* A past pericarditis does not prevent the characteristic signs of effusion occurring a second or a third time, provided there be not complete agglutination; friction-sound, pyramidal dulness, and twisting of the heart's apex upwards may all be present with, and through, the new attack.

1216. (*b*) In the second form, where agglutination of the serous surfaces and hypertrophy of the heart co-exist, each condition modifies the signs of the other. Thus the heart's apex may beat as high as natural in spite of the enlargement; if, however, the hypertrophy be very great, the apex falls, for the material of agglutination stretches. An unusual tendency to extension of percussion-dulness upwards is observed. Systolic dimpling is very marked; the lower end of the sternum and the adjacent left cartilages may be drawn in at the left costal angle. Jogging, trembling action of the heart may be highly marked.

1217. (*c*) The cardiac atrophy, distinctive of the third form, comes of tight embrace by the material of adhesions; especially, in all probability, if the coronary arteries be accidentally pressed upon. But such atrophy is of rare occurrence: a fact I ascribe to the rarity of thick strata of true induration-matter in chronic pericarditis.

The signs of simple adhesions will be conjoined with those of diminished size of the heart.

1218. *Treatment.*—The absorption of adhesions may be promoted by ioduretted applications, by mercurial inunction, and by the internal use of liquor potassæ and iodide and bromide of potassium. A course of the Woodhall or Kreuznach waters, internally and externally, is always worthy of trial.

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* Craddock, loc. cit.

If irritative action supervene, local depletion, blisters, caustic solution of iodine, with small quantities of mercury and digitalis or aconite internally, and free purgation, are the chief remedies. Subsequently the external use of belladonna and aconite will be found useful.

When dilated hypertrophy supervenes, the treatment must be essentially directed to that affection, on the principles elsewhere explained. And, certainly, the chief evil to contend with is generally hypertrophy and dilatation of the ventricles, mainly the left: where agglutination exists, this mode of enlargement of the heart almost invariably commences within a short period. But I confess that my observation does not lead me to take the very gloomy view expressed by some writers of the ultimate issue of such cases: I have not, as they appear to have done, seen mere chronic adhesive pericarditis, with hypertrophy, prove rapidly fatal. The question is, of course, a very different one if there be valvular disease superadded.

1219. (*d*) The symptoms and signs of fluid stagnating, chronically, in the pericardial sac are very closely those of the effusion-period of the acute disease.

1220. If these signs continue unchanged by ordinary means of treatment (hydragogue purgatives have little effect) paracentesis of the pericardium becomes justifiable, as an *ultima spes*, provided urgent suffocative symptoms exist. The patient is certainly not placed in a worse position by the operation, than he was before it; the immediate relief is extreme, and a certain very small chance exists of at least temporary recovery. Many years ago Professor Romero, of Huesca, published the results of eight cases, three without, five with, coexisting hydrothorax: in two of the former three the operation was successful.

If paracentesis be determined on (the danger of wounding the mammary arteries, the larger pericardial vessels, and the great arterial trunks being borne constantly in mind) the integuments should be incised opposite the central part of the heart in the natural state of things, that is, at the upper angle of the fourth left interspace [328], or a little lower than this; a trocar should then be introduced cautiously into the distended sac perpendicularly to the surface, the patient being in the recumbent position with the head moderately low.

The fluid, which escapes by jets, corresponding to the ventricular systoles, should be evacuated as completely as possible before the wound is dressed,—a syringe may even be employed to ensure this. But the orifice of the canula, before its removal, should occasionally be closed, lest too rapid abstraction of the fluid might produce evils of its own on the heart, accustomed as this has been, for a greater or less time, to considerable pressure. The practice of Romero, of allowing the fluid to gravitate into the pleural sac first, and thence outwards, seems unworthy of imitation. Some further information on this subject will be found under the head, Hæmopericardium [1268].

ACUTE ENDOCARDITIS.

1221. *Anatomical Characters.—In the membrane itself*, redness, florid in tint, distinctly vascular to the eye sometimes, and depending on injection of the vessels in the deep elastic layer and connecting tissue of the endocardium, according to Luscka, may exist pretty generally in patches, or sometimes more uniformly: or the hue may be pale dirty greyish yellow. The surface, deprived of its natural polish, feels more or less rough: the membrane becomes opaque, probably from filtration of exudation through the epithelium; grows thick, soft, velvety, and can be stripped or scraped off in short patches; and sometimes cracks, fibrinous coagula forming in the resulting fissures. *On the free surface*, patches of exudation-matter, at first gelatiniform and transparent, subsequently opalescent, form thin strata which may be peeled off, or small oval elevations,—the latter either on the general surface or on that of the valves or amid the tendinous cords. *Under the endocardium* sero-fibrinous exudation-matter forms in small quantities.

1222. Accidental characters are rupture of the tendinous cords, especially of the mitral valve, which then curl up and act as the nuclei of fibrine-coagulation; fissuring of the semilunar valves, with deposits of exudation and of fibrine directly from the blood; altered shape of the valves, and softening with rapid destruction of their substance.

1223. Ulceration, or at least destructive softening of the endocardium, may be followed by extension of the process beneath; an opening has thus been effected between the auricles. Pus sometimes forms in the muscular tissue, immediately beneath the endocardium; upon its free surface, in fissures; and, it is alleged, in the substance of fibrinous coagula, —softened fibrine has in the latter position, in some instances at least, been taken for pus.

1224. The formation of large coagula within the heart is frequent in severe cases: they bear all the acknowledged marks of production previously to death (their appearance can even be physically proved sometimes during life), are sometimes the seat of recent vascularisation, and are commonly softened in the central parts.

1225. The endocardium of the left side is much more frequently inflamed than that of the right; the inflammation may, however, be limited to the right; affect both sides; or mere fractions of one. The valvular apparatus is more commonly and more strikingly involved than the general tract of the membrane. Endocarditis of the right side in the fœtus has been suggested as a probable cause of coarctation of the pulmonary orifice, and hence of non-closure of the foramen ovale.

1226. Dr. John Taylor was the first to show, as matter of observation, that the products of endocarditis, washed away with the blood, might lay the ground-work of pulmonary, hepatic, splenic, and renal secondary inflammations [851].

1227. *Physical signs.*—In acute inflammation of the endocardium, the heart's movement is seen and felt to influence the surface more extensively, more forcibly, and more abruptly than natural. The cardiac region is not bulged, nor is the point of the apex-beat, as in pericardial effusion, raised upwards; it may be carried a little downwards and outwards even. There is no undulatory movement, and no tactile thrill: Hope says he has observed the thrill of mitral regurgitation, but he gives no proof that the regurgitation was purely recent; I have not succeeded in finding thrill, when any certainty existed of the absence of old-standing mitral disease. The areæ of the heart's dulness, both superficial and deep-seated, undergo increase; the former because greater energy of action brings the heart more uniformly forwards, the latter because the walls of the organ are turgid, and its cavities more or less clogged with blood. The area of dulness is never seriously increased, unless there be considerable distension of the heart by accumulated blood.

1228. Auscultation discovers a murmur or murmurs, blowing in quality, soft and low-pitched. The murmurs of *purely acute* endocarditis may, as far as I have observed, be thus arranged in order of frequency:—aortic obstructive; mitral regurgitant: aortic regurgitant; aortic obstructive and mitral regurgitant together; aortic obstructive and regurgitant together. Pulmonary systolic and diastolic murmurs are infinitely rare. I have never observed *acute* obstructive mitral murmur, nor *acute* regurgitant tricuspid murmur; the latter, especially, I believe to be at the least very rare,—a circumstance in accordance with the fact that most chronic tricuspid regurgitant murmurs are produced by simple incapacity of the valve to fill the widened orifice, without actual disease of its own tissue.

1229. The site and rhythm of acute endocarditic murmurs, it is supposed, may change during the course of an attack;—lymph, it is presumed, may be absorbed, or washed away and deposited elsewhere, and a different species of murmur consequently developed. I have not observed this; but I have known systolic aortic followed by diastolic aortic murmur, apparently from an increase of lymph.

1230. The murmurs of *acute* endocarditis are produced by roughness on the surfaces; by intertwined lymph, or mere fibrine, interfering with the play of the chordæ tendineæ; or, probably, by non-closure of orifices through irregular action of the papillary muscles.

1231. Such heart-sounds as are not replaced by murmurs, present no constant character. Reduplication of the second at the base is common. Probably, at the outset, they are both intensified; and murmurish prolongation of the first sound before it actually becomes a murmur, is sometimes noticeable both at the base and apex.

1232. In cases where the circulation through the heart's cavities is obstructed seriously, either from accumulation of lymph and fibrinous coagula, or from rupture of a valve or chorda tendinea, the impulse becomes irregular in force and rhythm,—at first violent, subsequently feeble; the heart's dulness notably extends, especially to the right of the sternum; the sounds or pre-existing murmurs are enfeebled, or new murmurs may be generated. The suddenness of occurrence of these signs points to their source.

1233. The *local symptoms* of endocarditis are not very marked. Pain is rare, discomfort and uneasiness at the heart common; more or less palpitation exists; tenderness of the præcordial interspaces is at the least unusual, unless there be co-existent pericarditis.

1234. The *general symptoms* vary with (*a*) the free, or (*b*) obstructed state of the circulation through the heart.

1235. (*a*) In the first class of cases the decumbency is generally dorsal, the attitude quiet; but jactitation of the arms occurs in some instances. The skin of febrile heat, the integuments unchanged in colour; the joints rheumatic, or unaffected; no special sensation of dyspnœa is complained of; the respiration holds its natural ratio to the pulse, so long as the orifices are not seriously obstructed, and no secondary pneumonia has occurred; sometimes a little dry cough exists without bronchial or other rhonchi; the pulse is not remarkably accelerated, ranging between 80 and 120,—Dr. Taylor's statement that it loses in frequency at the outset of endocarditis, I have not had an opportunity of confirming. The blood is hyperinotic in the sthenic disease; if the inflammation be the effect of phlebitis, pyohœmia, &c., the clot is soft, but little or at all buffed: endocarditis may also, in all probability, secondarily, cause the latter condition of the blood by the circulation of its own inflammation-products. The urine is simply febrile. Cephalalgia exists commonly more or less,—slight wandering may occur at night, but otherwise the head remains free. I once saw acute mania occur during the convalescence of endocarditis, of rheumatic origin, and unaccompanied with pericarditis.* Choreal symptoms are not induced, if the disease remain simple.

1236. (*b.*) In the obstructive class of cases, the action of the heart, suddenly at the moment of obstruction, becomes excessively frequent, uneven, and irregular; the pulse small, weak, irregular in force and rhythm, mounts to 130, 140, 160, or even more. Semi-syncope, pallor, coldness of surface, anxiety, and jactitation, inclination to orthopnœa (which the patient resists from its increasing faintness), with, eventually, the symptoms of complete pulmonary obstruction, lividity of surface, turgescence of the face, prominence of the eyeballs, puffiness of the ankles, supervene,—the brain suffers also congestively, as exhibited by fitful snatches of sleep, convulsions, delirium, and somnolence, lapsing into fatal coma. I have seen these symptoms in a minor degree, and passingly, in certain cases of endocarditis, which terminated favourably,—in all probability, in those instances, small concretions had formed, and subsequently undergone disintegration and solution. The symptoms of rupture of a chorda tendinea during the acute disease, are extremely similar; the effects on the cardiac circulation must, indeed, be closely analogous.

1237. The blood, in certain cases of endocarditis, receiving the products of the inflammation, undergoes alteration of composition. Rigors, heat of skin, and profuse perspiration recurring irregularly, dull, earthy, yellow discoloration of the skin, but not of the conjunctivæ, diarrhœa, more or less bilious, pinched, anxious countenance, intense prostration, and muttering delirium, subsequently cool clammy skin, and blueness of the finger-ends, announce this occurrence; and are followed by, or associated with, the evidences of secondary nodular pneumonia or hepatitis.

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* Cooper, U. C. H., Males, vol. i., p. 129.

1238. *Prognosis.*—The *terminations* of acute endocarditis are —(1.) Very serious valvular disease, followed by implication of the heart's substance, and all their combined consequences: the ensuing affections of the heart are, in their order of frequency, eccentric hypertrophy, simple hypertrophy, simple dilatation, and, in infinitely rare cases, eccentric dilatation.* (2.) Slight valvular disease, with habitual palpitation. (3.) Slight valvular disease, with palpitation under excitement. (4.) Simple murmurs, without any positive cardiac functional disturbance; no morbid palpitation occurring even under severe exercise: this is the most favourable result observed; an indubitable endocarditic murmur never, as far as I have known, totally disappears.† (5.) On the other hand, death is rare from acute endocarditis alone; still, the disease does occasionally kill, both by secondary impregnation of the blood, and by serious obstruction of the intra-cardiac circulation. Rheumatic endocarditis may run a perfectly *latent* course in regard of local and general subjective symptoms.

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* The relationship between the part of the heart affected and the diseased orifice, will be considered with valvular affections generally; for the endocarditic origin of the valve-disease gives no *special* character in this way.

† Temporary obscurity of a recent mitral murmur sometimes arises from the weakness of the heart attending convalescence, and leads to the idea that the murmur is gone, or will go, completely; as the general vigour improves, the murmur recovers its distinctness. Error is sometimes committed too in regard of this point, from change in position of the maximum site of murmurs, especially of the mitral regurgitant variety.

1239. Co-arising with endocarditis, or generated at least by the same influences, may be found rheumatic arthritis, pericarditis, carditis, aortitis, pneumonia and pleurisy. Aortitis is a much rarer concomitant, at least as far as proof goes, than might be expected. Bright's disease, rheumatism, gout, and pyohæmia, are the only diathetic affections, that clearly promote the occurrence of endocarditis. Neighbouring inflammations, old cardiac disease, injuries to the præcordial region and violent efforts are sometimes traceable as its causes. The existence of old valvular disease renders the endocardium more prone to acute inflammation.

Diagnosis.—The diagnosis of acute endocarditis is essentially based on the existence of febrile action, cardiac uneasiness, excited action of the heart, and endocardial murmur,—this murmur occurring in a person presumedly free from prior cardiac disease, and presenting no other obvious acute affection to explain the febrile action.

1240. But a murmur, existing when a patient is first examined, may be new, or it may be old. Now granting that it is new, it may not be the product of endocarditis. For the excitement of the heart may depend on some other, latent, inflammation or as yet unevolved disease; and this excitement, coupled with a modified state of the blood, may suffice to generate a murmur. Thus, occasionally, at the outset of pneumonia, of the exanthemata, and of any inflammatory state, in persons whose blood chances to be in the least spanæmic, murmur occurs. But such murmur, purely of blood-origin, is necessarily systolic and basic: if there be new murmur, either systolic or diastolic at the apex, or diastolic at the base, a complication of acute endocarditis must be admitted; if the murmur be basic and systolic only, it may be hæmic, or it may be endocarditic, and consequently the diagnosis must be deferred. Besides, deposition of fibrine may take place acutely amid the cords, or at the edges of the valves, and induce murmur, without the intervention of inflammation.

1241. So far the murmur has, for argument' sake, been admitted to be new; unfortunately the great difficulty is often to determine that it *is* new. Now two cases present themselves here: (*a*) the murmur exists, when the patient is first seen; or (*b*) it is developed, after observation of him has commenced.

1242. (*a*) A murmur being already present, the circumstances, *within itself*, favourable to recency of origin, are softness of blowing quality, lowness of pitch, systolic rhythm and aortic constrictive or mitral regurgitant mechanism. The circumstances hostile to recent origin are roughness of quality, high pitch, diastolic rhythm (indeed this is absolutely conclusive, unless there be systolic murmur at the same orifice) and seat at the tricuspid orifice; direct mitral murmur, also, I believe, is never recent. The presence or absence of affections with which endocarditis is commonly associated, furnishes a guide not to be despised; but without caution the observer may readily be led into error, as in acute rheumatism, by too implicit trust in this very guide. The condition of the pulse cannot be confided in for diagnosis.

1243. (*b*) An endocardial murmur, *developed under observation, at the early period of an acute attack*, is almost a sure index of endocarditis; but even here there are sources of fallacy. In the first place appear those just enumerated, on the hypothesis of the murmur being by admission new, and yet of hæmic origin. In the second place, general collapse and failure of the heart's power may, on the first examination of a patient, prevent a given murmur, of which the physical chronic conditions exist in perfection, from being heard. Reaction takes place, and a murmur becomes audible; that murmur may be chronic murmur solely, or it may be an acute and a chronic murmur combined; but it is *not* that, which it would alone seem to be, namely, an acute murmur *solely*.* Again, to have value as a positive sign of recent endocarditis, the murmur must be developed at an *early* period of acute disease; if towards the close, it is almost invariably systolic in time, basic in seat, and a consequence of spanæmia. Further, the possible occurrence of murmurs of dynamic mechanism, muscular and valvular, must not be forgotten. Not only in chorea, but in other nervous diseases, and in nervous conditions supervening in the course of acute maladies in general, is their occurrence a possibility, nay a probability. Such dynamic murmurs are generally systolic and seated at the apex [442]; but there is much reason to suppose that murmurs may also occur dynamically at the base from perverted action of the sygmoid valves.

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* Kernis, U, C. H., Females, vol. ii., p. 237. The statements in the text are made on clinical and *post-mortem* evidence.

1244. If during the course of an acute febrile disease, endocardial murmur changes in site and rhythm, this is a very strong, though not an absolute, sign of its dependence on recent inflammation; and, admitting its value, such change is, we have seen, at the least very rare. Again, if a murmur of a certain site and rhythm disappear and return within a short period, the changes might appear proof positive of recency of the cause of the original murmur; but it is not so, at least of murmurs of all varieties of localisation. Thus, I have known a direct mitral murmur, essentially organic and attended with great constriction of the orifice, come and go from one day to another [449]: some dynamic conditions must now have obstructed, now have promoted, the occurrence of the murmur.

1245. And there is yet another possible cause of error: an endocardial murmur appears sometimes to occur, according to the testimony of Drs. Latham and Stokes, shortly before death from the impediment of coagulated blood. Fortunately this *præ-mortem* murmur is of rare occurrence: how, if at all, it may be distinguished from an endocarditic one, remains to be ascertained.

1246. So far intra-cardiac murmur has been assumed to be unfailingly present. But may it not be absent in acute endocarditis? The conditions producing murmur are roughness of valves, lymph among the tendinous cords, insufficiency of valves from puckering, and notable roughness, from lymph, of the ventricular surface: now as endocarditis may exist without any one of these anatomical effects, the inference is unavoidable, that it may occur without murmur. I have seen distinct patchy redness with thin films of lymph on the ventricular endocardium, where there had been recent excitement of the heart without newly-developed murmur.*

1247. Endocarditis will be distinguishable from pericarditis by the less amount of pain, by uneasiness not being increased by a deep breath, cough or change of posture, by absence of præcordial tenderness, by the less amount of oppressive breathing, and by the general difference of the physical signs. The distinctions of endocardial and pericardial murmurs will be found elsewhere [474, 1198].

The formation of polypoid concretions may be very strongly suspected if, with the *sudden* appearance of the symptoms enumerated above [1236], a notable increase in the area of percussion-dulness be detected. But rupture of a sygmoid valve, or of a tendinous cord, will produce closely the same effects.

1248. *Treatment.*—The treatment of endocarditis is essentially the same as of pericarditis, and active measures are obviously called for, as the ultimate dangers of valvular disease are extreme. Blood-letting and mercurials are the main agents.

1249. When the affection seems lapsing into the chronic state, iodide of potassium and liquor potassæ, with bitter tonics, become the best remedies; and iodine-inunctions over the cardiac region appear occasionally useful.

1250. If there be reason to suspect the formation of polypoid concretions, the rapid pouring in of liquor-potassæ seems, theoetically at least, worth trial; if there be sinking tendency, esquicarbonate of ammonia may be given at the same time. inapisms should be applied to the extremities. I have seen emporary relief obtained by cupping over the heart: but the atient's strength is rarely, when such obstruction occurs, in a ondition to bear the loss of even a few ounces of blood; and the pplication of Junod's apparatus is a safer measure.

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* "Shreddy lymph on surface of left columnæ carneæ recent," without change in character of pre-existing chronic murmurs. Kernis, U. C. H., Females, vol. ii., p. 244. March, 1847.

CHRONIC ENDOCARDITIS.

1251. Chronic endocarditis is signified by thickening and pacity of the membrane,* uniformly or patchwise, perfectly mooth or slightly and irregularly puckered. Successive strata, hin, opaque, faintly yellow, and filmy, may be peeled off from he free surface; or minute masses of hard, elevated, yellowish nduration-matter separated with the point of the scalpel, leaving he endocardium, apparently uninjured, beneath.

The valves suffer variously. Thickening by infiltrated induraon-matter, with hardness, opacity and puckering of their subtance, sometimes associated with diminished depth from acute estruction or chronic contraction, are the most common ppearances. Calcification, or pseudo-ossification, may ensue in atches, nodules, or spiculæ. Adhesion of the divisions of a alve to each other, or to the neighbouring surface, is sometimes een: the former, most common in the aortic valves, may cause hem to pouch in the wrong direction; but such adhesion, conerting them into a single indurated ring, occurs to perfection in he mitral valves also. Thickening and shortening of the mitral ords, sometimes conjoined with special thickening of the endoardium of the papillary muscles, is clinically one of the most nportant of chronic changes.

Warty fibrinous products, polypoid or sessile, soft, firm, or alcified, form on the valvular surfaces exposed to the blood-current. The free edge of the mitral, less frequently of the tricuspid valve, frequently presents a semi-transparent beaded thickening, which is not clearly inflammatory, or even morbid.

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* The student must not forget the natural excess of opacity and thickness, rticularly as age advances, in the endocardium of the left auricle.

1252. *Symptoms.*—Although, very possibly, certain uneasy sensations about the heart, and proneness to palpitation, may depend upon chronic changes in the ventricular or auricular endocardium, yet there is no surety of knowledge on the point. Hence, in point of fact, chronic endocarditis is solely known clinically by its effects on the valves and orifices of the heart; and there is nothing in the physical or dynamic characters of valve-disease originating in inflammation, distinguishing it from that produced by other causes. For the clinical history of chronic endocarditis the reader is consequently referred to the section on valvular affections in general.

ENDO-PERICARDITIS.

1253. Inflammation often attacks both membranes of the heart, either coetaneously or consecutively: rheumatic endo-pericarditis is, if Dr. Latham's returns be accepted, somewhat more frequent than pericarditis alone, greatly less frequent than endocarditis. But it is difficult to resist the conviction that the great excess of endocarditic cases in part depended on old intra-cardial murmurs being classed as new; especially as the results of other observers do not tally with those of Dr. Latham.

1254. The friction-sound of pericarditis often renders the detection of existing endocardial murmur difficult or impossible; a large amount of pericardial fluid also commonly weakens, while it may very possibly create [437], endocardial murmur. The signs and effects of endocarditis on the other hand cause no difficulty in the discovery of pericarditic signs. In other words, pericarditis obviously existing, endocarditis may very well be present, though its signs are imperfectly discoverable; whereas the converse does not hold good.

1255. The prognosis of the double inflammation is more serious than that of either singly.

ACUTE CARDITIS.

1256. I. The anatomical characters of acute carditis, as an attendant on endo-pericarditis, are well known; they are of olerably frequent occurrence, on a limited scale, in the strata of ibres nearest the inflamed membranes, especially the pericardium. I have seen exudation-corpuscles and pus-cells amid the ibres under these circumstances. But clinically, the effects of such carditis are not understood; whatever they are, they are ost in the more striking phenomena of the membranous inflammations; possibly great weakness and fluttering character of he pulse may sometimes be due to inflammatory softening of he left ventricle, in cases of pericarditis without much fluid effusion.

1257. II. General inflammation of the heart's substance is very rare. Vascular injection, deepened colour, with crispness, not real firmness, of the texture, seem its earliest characters. Exudation, sero-sanguineous, or of lymph, or of pus either in the form of infiltration or abscess, ensues; the texture softens even to pulpiness, and, commonly of pale yellowish or greyish brown hue, may, if there be much infiltration of blood, acquire a blackish int. If there be local softening and abscess, the part affected may bulge outwards, and actually give way into the pericardium. Ulceration extending a certain depth into the wall of a ventricle, or perforating either the pericardium, or the ventricular, or auricular septum has rarely, gangrene still more rarely, been seen. The left ventricle is the most frequently affected. Of the influence of age nothing is known; males are the most common sufferers.

The pericardium seems to be almost always secondarily affected; the endocardium often escapes,—a fact the more strange, as there is strong ground for believing that carditis originates in some special poisoned state of the blood, and is never truly a primary idiopathic disease.

1258. The narratives of the few cases of carditis on record furnish no direct clue to the detection of the disease. Some were observed before the days of physical diagnosis; others, in which percussion and auscultation were performed, presented, it is averred, no sign. The heart-disease has been thrown into the shade by the predominance of cerebral and other symptoms, indicating some form of poisoning of the blood, allied to or identical with pyohæmia.

In point of fact the disease has never been diagnosticated, that I know of, and has to be *observed*, before its clinical description can be written.

1259. III. Partial carditis sometimes occurs, producing abscess, ulceration, and rarely actual gangrene; but of these states no positive clinical signs are known. If perforation or rupture occur, as they sometimes do, the symptoms will vary with its direction; if the septa be perforated, sudden cyanosis may follow, or, it is alleged by Bouillaud, no particularly serious symptoms ensue. The effects of rupture of the heart into the pericardium will be elsewhere considered.

1260. IV. Minute collections of pus form in the heart's substance in a certain proportion of cases of pyohæmia and phlebitis,—greatly less frequently however than in the lungs and liver. The symptoms are yet to be worked out.

CHRONIC CARDITIS.

1261. Shortening with thickening of the papillary muscles and columnæ carneæ, and infiltration of their substance with induration-matter, due to a chronic inflammatory process, mainly derives interest from its interference with the closure of the mitral, in very rare instances of the tricuspid, valves; but there is no character in either a mitral, tricuspid, or regurgitant murmur, distinctive of this special mechanism.

CARDIAC HÆMORRHAGES.

1262. I. *Of the Muscular Substance.*—Hæmorrhage in this site may form specks, or apoplectiform nodules of blood: or ppear in the infiltrated state in connection with (rather, erhaps, as effect than cause) local softening. Under the latter ircumstances, rupture or perforation of the heart may occur, nd produce fatal extravasation of blood into the pericardium. uch mode of death might be suspected in an individual uddenly cut off with signs of accumulation in the pericarlium, and previously known to have a weak heart and to be ree from aneurism of the aorta: but there are no positive ymptoms of hæmorrhage limited to the heart's actual subtance.

1263. II. *Of the Pericardium.*—(*a.*) The effusion of inflamnation is sometimes so much stained with blood, as to entitle he disease to the name of hæmorrhagic pericarditis. It seems robable that such escape of the blood-disks does not take place rom the vessels, unless the constitution of the blood itself be ffected. I have seen this variety of inflammation well marked n pyohæmia. But no signs or symptoms are known, whereby he hæmorrhagic addition to the effusion might be recognised uring life. Neither is there any evidence to show that, in the vent of recovery, the material of adhesion will be of different haracter from that observed in the more ordinary class of cases. he quantity of blood is sometimes very considerable,—quite nough to give a deep red colour to all the fluid in the sac: ere paracentesis performed in such a case, the operator would ery probably be led, for a moment, to believe that he had unctured some important vessel.

1264. (*b.*) Hæmopericardium may be caused by wounds or y ruptures of the heart itself, by rupture of an aneurism of the orta or of the heart, by rupture of a coronary artery or vein, or y the giving way of cancerous substance. In all these cases he result is almost instantaneous death, from mechanical bstruction of the heart's action. Some of these cases will gain be referred to; the rest are devoid of clinical interest.

1265. (*c.*) The pericardium is the seat of extravasation of lood in cases of scurvy,—especially in some localities; in certain parts of Russia, scorbutic hæmopericardium seems as thoroughly endemic as hæmaturia in the Mauritius.

1266. It occurs with or without previous scorbutic symptoms, and the attack may be sudden, or so gradual that attention is scarcely drawn to the heart. Præcordial oppression, without pain, or tenderness, and great dyspnœa, seem to constitute the main symptoms; the physical signs will, of course, be those of a pericardium distended with fluid.

1267. The more frequent termination is by death; but recovery sometimes takes place by absorption of the blood,—the anatomical conditions remaining, being very assimilable to those of chronic pericarditis. Indeed, it seems highly probable that the disease is from the first sub-inflammatory.

1268. The treatment, locally, is by cupping,—constitutionally, that of the blood-disease present. In the majority of a number of cases, in which paracentesis was performed by Russian physicians, the ultimate result was unfavourable; but in all the immediate relief was extreme,—the patient seemed temporarily endowed with new life: and two cases are given of complete recovery. In one of these, related by M. Karawajew, three pints and a-half are stated to have been removed from the pericardium; pneumo-pericardium followed; five months after the operation the patient was considered well.—("Brit. and For. Med. Rev.," July, 1841.)

HYDROPERICARDIUM.

1269. Hydropericardium, or dropsy of the pericardium, may be of active, passive, or mechanical origin.

1270. (*a.*) Active hydropericardium is very rare: I have, however, in some instances of Bright's disease, known the pericardium fill with fluid,—the symptoms indicating an irritative state, while the signs of pericarditis were wanting. I once saw a case which suggested to my mind the question, whether in true hydropericarditis, the plastic material might not be completely absorbed, and the serosity left behind,—constituting a sort of sequential active hydropericardium. But I have no positive answer to supply; and, possibly, the case referred to was one of active dropsy alone from the first. When hydropericardium is active, it may be the sole dropsy in the body.

1271. (*b*) Passive hydropericardium occurs as a phenomenon of general dropsy,—very rarely unless double hydrothorax be already present. The quantity of fluid (colourless, straw-coloured, or slightly blood-stained, but without lymph) is generally moderate,—from eight to twelve ounces; I have never seen more than the latter quantity.*

1272. (*c*) Mechanical hydropericardium has, in some very rare cases, been traced to pressure of carcinoma on the great veins, and to morbid states of the coronary veins.

1273. The mechanism of hydropericardium is sometimes very difficult of explanation. Thus in a phthisical male, aged twenty-four, cut off in about four hours by perforation of the right pleura and pneumothorax, with little pain, but intense dyspnœa and general distress, the pericardium, pyramidally distended up to the second rib, was found to contain nine ounces and a half of clear faintly yellowish serosity, of specific gravity 1013 and neutral reaction, giving one third albumen on boiling: there was not the slightest vascularity of, or lymph upon, the serous membrane, which simply looked macerated.† The lung was flattened against the side, but sufficient time had not elapsed for the occurrence of pleuritic changes. Could this hydropericardium have been the result of the sudden pressure of, and obstructed circulation in, the right lung? It is true, hydropericardium does not appear in the narratives of cases of phthisical perforation; but death rarely takes place so rapidly as in this instance,—and possibly after the first shock of the mechanical difficulty is past, absorption may commence.

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* A case recorded by Corvisart (Maladies du Cœur, 2ème édit., p. 52), where "about four pints, or eight pounds, of clear greenish serosity," were found in the pericardium, seems to have been one of chronic pericarditis. The distension, which the sac must have undergone, is the more remarkable as the membrane is stated to have been thickened.

† Imlach, U. C. H., Males, vol. viii. p. 402.

1274. Hydropericardium, however originating, has few subjective symptoms: acute pain and tenderness are altogether, and palpitation commonly, wanting; sensations of weight and oppression are alone complained of. The physical signs are in the main those of hydropericarditis: but there is no friction-sound, no præcordial bulging, and the apex of the triangular dulness (mainly because there is less fluid, but partly because there is no inflammatory relaxation and rarefaction of the sero-fibrous membrane) does not mount so high as in many cases of pericarditis,—the impulse may be very perfectly undulatory.

1275. The general symptoms are those of the disease on which the local dropsy depends. The pulse is not necessarily irregular, the quantity of fluid being commonly insufficient to affect the heart's action very seriously; while the constrictive influence of exudation-matter, and the dynamic perversions of muscular action through contiguous inflammation, are altogether wanting. Orthopnœa may exist; but if so, as far as I have seen, there is double hydrothorax to share in its production.

1276. *Treatment.*—Diuretics and hydrogogues seem to have less effect on this dropsy than on others: such medicines will, however, of necessity, be tried, were it only for the usually concomitant dropsies. Cautious cupping, or dry-cupping over the heart, would be advisable, if the symptoms became urgent. A blister has sometimes appeared to me useful. Paracentesis has been performed, with temporary relief; but unless the primary disease be removed, of course the fluid will be reproduced. I should scarcely like to adopt the suggestion of Laennec, and inject slightly irritant fluids into the pericardium, so as to excite an inflammation that might, by causing adhesion, prevent the recurrence of the disease.

ŒDEMA OF THE HEART.

1277. I have seen the heart's substance of watery look, and distinctly infiltrated with serosity, both in cases of chronic pericarditis of the persistent effusion-form [1219], and in cases of general dropsy. Ill provided with cellular tissue, like the brain, the heart rarely becomes œdematous to any extent. In chronic pericarditis, it is rather the intervening texture between the heart and its membranes, than its own substance, that undergoes infiltration.

1278. In the present state of knowledge these conditions are merely anatomically interesting. Yet it seems probable that œdema, carried to any amount, may interfere with the heart's contractions.

1279. Serous infiltration under the endocardium and between the layers of the sygmoid and of the mitral valves has also fallen under my notice; but I know nothing of either state clinically.

PNEUMO-PERICARDIUM AND PNEUMO-HYDROPERICARDIUM.

1280. Theoretically described by some authors, these affections have never been observed, much less diagnosticated, during life. It is excessively doubtful whether the one or the other ever occurs as an idiopathic state.

1281. Pneumo-pericardium must exist temporarily as the result of perforative communication between the pericardial sac and any hollow viscus containing gas; but in this isolated state it has never been observed,—pericarditis having supervened before clinical examination has been made.

PNEUMO-PERICARDITIS.

1282. Gaseous fluid may conceivably accumulate in an inflamed pericardium as an actual formation by the irritated membrane; it may result from decomposition of liquid effusion; or be conveyed into the sac by perforation, traumatic or diseased, from a neighbouring hollow canal or viscus.

1283. (*a*) Dr. Stokes briefly describes a case of pericarditis, in which for three days metallic guggling and crepitating sounds were heard over the cardiac region in accompaniment with amphoric and cracked-metal note on percussion,—the stomach not being distended with air and the lungs and pleura unaffected. The heart's sounds were audible at a distance. It seems equally difficult to contest the dependence of these phenomena on the presence of air in the sac, and to explain such pneumatosis by acknowledged pathological laws. There was no *post-mortem* examination.

1284. (*b*) The fluid of pericarditis, fœtid and decomposed, has been known to furnish gas during life. In a case of this sort, observed by M. Bricheteau, the heart's action was accompanied with a "sound like that of a water-wheel." The actual presence of gas in the sac was ascertained after death.

1285. (*c*) In the singular case of traumatic communication between the œsophagus and pericardial sac, more than once referred to [378], the percussion-resonance over the heart, when I examined the case, was purely tympanitic, not in the least tubular or amphoric; no guggling sound accompanied the heart's action, nor were its sounds, or the existing friction-sound, particularly loud. The distinctive phenomenon consisted in the change of position of tympanitic and dull percussion-sound, within the area of the cardiac region, according as the posture of the patient was changed from one to the other side.

1286. Dr. Graves records a case of fistulous communication between a hepatic abscess and the stomach on the one hand and the pericardial sac on the other. Loud metallic ticking with each stroke of the heart, combined with friction-sounds and a noise like emphysematous crackling, were the signs of pneumo-pericarditis following perforation. Dr. M'Dowel has observed a case of communication between a cavity in the left lung and the pericardium: metallic tinkling, amphoric buzzing, and splashing of fluid were caused by the action of the heart.

ATROPHY.

1287. *Valvular Atrophy.*—When the cordæ tendineæ of the mitral valve are shortened and extremely thin, they are, probably, purely atrophous. The larger tongue of this valve is sometimes simply defective in size, without obvious puckering, or other evidence of past inflammation. In both cases, regurgitation may occur. Reticulation of the mitral valve is rare.

1288. The sigmoid valves may be thin and papery,—whence a sharp clicking state of the second sound, but no actual disturbance of the heart's action. These valves, too, may be reticulated or cribriform,* a state conceivably the cause of regurgitant basic murmur. But I have never known a murmur actually so produced; and if its occurrence were usual, the murmur signifying pulmonary regurgitation ought, instead of being one of the *mirabilia* of clinical practice, to be common, —seeing that reticulation is very closely as frequent in the pulmonary, as in the aortic, valves. Besides, there is an anatomical cause why, unless in extreme cases, reticulation should have no disturbing influence on the circulation; it affects those parts of the valves, close to their free edges, that lie surface to surface in the centre of the vessel, at the moment of its systole;—these particular portions of the valves have nothing directly to do, physiologically, with the prevention of regurgitation [451].

1289. The tricuspid valve is sometimes at once deficient in depth, its substance thin and papery, and its cords excessively delicate,—conditions apparently proving deficient nourishment.

1290. *Heart.*—(*a*) The heart is said to be the subject of *concentric atrophy*, when the size and weight of the organ and the capacity of its cavities are alike diminished. In various degrees this form of atrophy occurs in wasting diseases, cancer especially; it appears sometimes to follow the tight embrace of pericardial false-membrane; to depend occasionally on narrowing and calcification of the coronary arteries; and, it is alleged, has sometimes been artificially produced by the means adapted for the treatment of hypertrophy,—a statement which, although emanating from Laennec, requires corroboration.

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* I speak of this state under the head of atrophy, to avoid multiplying divisions: it is very doubtful whether it is atrophous; neither do I think Bizot's theory (of rupture by extension from rapid growth) at all satisfactory. —(Mém. de la Soc. Méd. d'Obs. de Paris, tom. i., p. 367.)

1291. No local symptoms are positively traceable to this state; palpitation has sometimes been observed, but (as in advanced carcinoma) is, probably, rather the result of spanæmia, than of the atrophy. The impulse is deficient in force and extent,—the area of percussion-dulness lessened; of the characters of the sounds, I know nothing of any importance. The pulse is small.

1292. (*b*) *Eccentric atrophy.*—When the walls of the heart are greatly attenuated with or without subsidiary dilatation, loss of mass has occurred, and the state may fairly be termed one of eccentric atrophy. It is singularly rare; and is rarer still in the left than the right ventricle. In the latter situation it probably intensifies some of the effects of the dilatation and tricuspid insufficiency, with which it is commonly associated; but, in point of fact, I have seldom seen atrophy of the kind.

HYPERTROPHY.

1293. § I. *Valvular Hypertrophy.*—Thickening of the valves is sometimes observed in connection with hypertrophy of the left ventricle, when no anatomical or clinical indications exist of bygone inflammation; and where, in all probability, the thickening results from extra-nutrition consequent on the extra work entailed on the valves by the muscular hypertrophy. This state of the valves gives a dull, heavy, clanging character to the valvular portion of the first, and especially, to the second, sound; but is rarely, if ever, carried far enough to produce either obstructive or regurgitant murmur.

1294. § II. *Cardiac Hypertrophy.*—Hypertrophy of the muscular substance of the heart may be *simple*, the affected cavity and its walls retaining their natural relative proportions; or *eccentric*, with dilatation of the cavities; or *concentric*, with contraction of the cavities. Hypertrophy may be general, in which case it is almost invariably eccentric; or, limited to a single compartment of the heart,—under which circumstances, the species varies. The left ventricle is the most frequent seat by far of the disease; next comes the right ventricle; then the left, and lastly the right, auricle.

1295. The most common form of the disease seems to be hypertrophy with dilatation of both sides of the heart, but carried to much greater amount on the left than the right side. In extreme cases the bulk of the organ may be augmented to double, treble, it is even said quadruple the natural size; and its weight increased to two pounds or even upwards. The heart's form becomes unnaturally spherical, square, or it may be broader than long; the apex cannot be distinguished. Habitually depressed, the organ may be retained in the natural position by pleuro-pericardial adhesions,—and may even reach to the second interspace above and extend below to the eighth rib, stretching vertico-diagonally from nearly two inches to the right of the sternum, to two and three quarter inches outside the line of the left nipple. The aortic orifice widens, so as to exceed the pulmonary in width. The coronary arteries, as also the nerves and gangliform swellings (See Cloetta), enlarge.

1296. The characters of the fibre in hypertrophy appear to be those of health; and as the primitive fasciculi do not increase in thickness, new ones are obviously formed. Production of non-striated fibre, or even of fibre more imperfectly striated than in health, does not occur. Firm hypertrophy in one part of the walls of a ventricle may be associated with softness and anæmic tint, or with fatty change, in another.

1297. The causes of general or limited hypertrophy of the heart may be set down as follows: in some instances the reality of the assumed influence is doubtful,—a fact signified in each instance by a note of interrogation.

I. *Causes originating in the system at large.*—Excessive nourishment, especially nitrogenized, combined with free use of stimulants, and sedentary habits; excessive exercise, as pedestrian, rowing (and that of trades straining the upper extremities in excess?).

II. *Causes originating in the blood.*—Uræmia? rheumatic hyperinosis?

III. *Causes originating in the heart itself.*—(*a*) *Functional:* Excitement of the heart, as the habitual palpitation of prolonged anæmia; irritation of chronic pericarditis [1204]. (*b*) *Mechanical obstruction:* (1.) *Affecting left ventricle:* Aortic constriction, aortic regurgitation, mitral regurgitation, mitral constriction?* mitral regurgitation and constriction combined. (2.) *Affecting right ventricle:* Tricuspid regurgitation, pulmonary constriction, mitral regurgitation indirectly through engorgement of the lungs. (3.) *Affecting left auricle:* Mitral regurgitation and constriction, aortic regurgitation and constriction in much less degree. (4.) *Affecting right auricle:* Tricuspid regurgitation.

IV. *Causes originating in the great vessels.*—Pressure obstructing their interior; smallness or constriction of aorta; aneurism of aortic arch near the heart; diminished elasticity of coats of aorta or pulmonary artery, affecting severally left or right ventricle?

V. *Causes originating in the lung-circulation.*—*Affecting right ventricle:* Chronic bronchitis? emphysema; contraction after pleurisy; dilatation of bronchi and cirrhosis of the lung; diminution of cavity of chest, by deformity or by pressure of abdominal tumors? It is possible too that the left ventricle may eventually become hypertrophous, from the strain thrown on it by the systemic, sequential to the pulmonary, obstruction?

VI. *Causes originating in the kidney.*—Persistent obstruction in its capillary circulation??

1298. The symptoms, signs, and effects of hypertrophy of the different compartments of the heart differ so materially, that, to avoid confusion, it will be advisable to consider the disease in each situation separately. Nor must it be forgotten

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[*] Mitral constriction may be hypothetically supposed to cause left ventricular hypertrophy, either through the effort of the ventricle to overcome the systolic capillary obstruction consequent on the pulmonary obstruction, immediately induced by the mitral constriction; or through the extra effort made by the ventricle to propel the small quantity of blood supplied by the auricle, to make up, as it were, by force of propulsion, for smallness of supply.

that pure and simple examples of hypertrophy are rare; that valvular affections commonly exist to modify the results.

1299. I. *Hypertrophy of the general substance of the Left Ventricle,*—either pure, or combined with dilatation of the cavity insufficient in amount to throw its own special characters into the shade. In the following description a highly-marked case is taken as the model.

1300. *Physical signs.*—Inspection discloses arching of the præcordial region (especially in long-standing cases, and in early youth), with widening, but without bulging, of the left interspaces, from the third to the seventh. The impulse, increased in extent, especially to the left of the sternum, presents its maximum amount below and about the left nipple, and between this and the sternum; in character it is slow, heaving, and suggestive of pressure forwards steadily against an obstacle; in rhythm regular, unless there be some added morbid state; in force unequal. The amount of force may be sufficient to shake the head of the observer, the trunk of the patient, or the bed even on which he lies; such extreme power of action is rare, unless dilatation be combined with great hypertrophy. Of double systolic and diastolic impulse, I have already spoken [360]. The point of the apex-beat, carried downwards and outwards, may reach the lower edge of the seventh rib (rarely, however, without dilatation), at some distance outside a line let fall perpendicularly from the nipple. In eccentric hypertrophy the extent of visible impulse is much greater; the apex-point may be carried to the seventh space or eighth rib; the impulse may, without much difficulty, be felt in the back; its character is less heaving than in the pure disease, sharper, more knocking, or slapping, and the surface, over which it is perceptible to the hand, proportionally more extensive.

1301. The superficial and deep-seated dulnesses of the heart are both augmented in area, and, probably, in amount also,—the parietal resistance is sometimes very notably increased. In dilated hypertrophy, the dulness may reach from the second interspace (here some slight increase upwards has occurred) to the eighth rib, and from an inch and a half to the right of the sternum to three inches and even upwards outside the vertical line of the nipple: and dulness may be detected in the back to the left of the spine. The rudely triangular form, natural to the heart's *superficial* dulness (*vide* Diagram II., p. 175), gives place to a dulness of somewhat square outline; and the deep-seated dulness is also more right-angled than in health;—this latter character, however, is not often to be satisfactorily ascertained.

1302. In simple hypertrophy, the first sound is dull, muffled, prolonged, weakened in some cases almost to actual extinction, directly over the ventricle, the sensation reaching the observer's ear being rather one of impulsive motion than of sound;—under these circumstances a tolerably full systolic sound may, nevertheless, frequently be found at the base and at the ensiform cartilage; the extent of its transmission towards the apex is very limited. The second sound, though wanting in clearness, may be full and clanging; the post-systolic silence is shortened. During palpitation the first sound sometimes becomes comparatively clear. In hypertrophy with dilatation, the sounds gain greatly in loudness, and extent of transmission, especially if the valves be perfectly healthy and free even from hypertrophous thickening; and the tone of the first at the left apex is notably clearer than natural. Reduplication of either sound, sometimes occurring, possesses no special character: it is not common,—especially when the inequality of strength in the two ventricles is considered.

1303. Systolic blowing murmur, basic and audible at the second right cartilage, is sometimes heard in cases of pure hypertrophy; nor can it be positively ascribed in all instances to coexistent spanæmia. I have known such a murmur disappear, when the heart had become comparatively quiescent by treatment of a depressing kind rather than otherwise, and wholly non-ferruginous. Hence excess of force of propulsion of naturally constituted blood would seem capable of generating *direct* murmur. Hypertrophy may possibly, during the excitement of palpitation, induce mitral regurgitant murmur by disturbing the action of the papillary muscles,—especially as these muscles are often relatively nourished more, sometimes less, than the rest of the ventricle. Such murmur actually does, as a clinical fact, exist at one time and disappear at others. And systolic basic murmur may also, very probably, be generated in cases of dilated hypertrophy, in consequence of the altered relationship of the aortic orifice to the cavity of the ventricle—altered both in point of size and of direction of the blood-current [436, 442]. In dilated hypertrophy, knocking and rubbing additions to the first sound at the apex, either left or right, are not very uncommon. Possibly, too, the second sound may be, in seeming at least, intensified at the left apex by the abrupt recedence of the enlarged heart from the side during its diastole.

1304. The respiration at the centre of the cardiac region, that is, the upper sterno-costal angle of the fourth left interspace, is feeble and distant,—but not so feeble nor so distant as it would be with an equal amount of percussion-dulness from fluid in the pericardium.

1305. In estimating the dulness really depending on an hypertrophous heart, the observer must bear in mind, that its apparent extent may be, on the one hand, increased by engorgement of the right cavities, from temporary disturbance of the circulation, by aneurism of the aorta, by indurations in the lungs, pleura, or mediastina, by tumors of the œsophagus even, and by enlargements of the liver, which, by pushing the organ upwards and to the left, widen the area of dulness in those directions;—and, on the other hand, decreased by emphysema and bronchitic distension of the lung.

1306. *Symptoms and state of the functions generally.*—The state of the functions may be described as follows, in cases of pure hypertrophy, or hypertrophy without any notable amount of dilatation. (*a*) The strength does not seriously suffer, unless the disease be carried to a great height; the power of walking and of ascending hilly ground is diminished, not from feebleness, but from the dyspnœa and oppression induced by the attempt. Patients generally lie with the head high. (*b*) The colour of the integuments varies; if the hypertrophy be pure, the face is florid, if coupled with moderate dilatation, there may be slight purpleness and lividity; but marked purple discoloration does not occur, unless there be very considerable dilatation, valvular obstruction, or pulmonary disease. Hypertrophy of the left ventricle does not *per se* produce œdema of the ankles, much less general anasarca: even hypertrophy and dilatation, unless the latter be in great excess, fails to induce this evidence of systemic vascular obstruction [1326]. (*c*) The muscles are well nourished and of good colour. (*d*) There is not any form of dyspeptic derangement particularly assignable to hypertrophy; the disease may exist for years without materially affecting the digestive powers, provided moderate exercise be taken. Constipation acts as a source of habitual annoyance. (*e*) There is more or less dyspnœa, either constant or occasional,—in the latter case induced by the most trifling effort: the pulse and respiration-ratio may be perverted in consequence; paroxysms of dyspnœa have not occurred under my observation, unless there were much dilatation, valvular obstruction, or pulmonary disease. Dry cough annoys some patients; I have not observed œdema of the lungs. (*f*) The radial pulse, in no wise peculiar in regard of frequency, and perfectly regular in rhythm, is full, strong, firm, tense, resisting, and prolonged, without jerk or thrill, in the pure disease; if dilatation be superadded, it retains its fulness, but loses in some measure strength and resisting power. It is said not to be increased in frequency, as in health, by change from recumbency to the sitting and standing postures. The action of the carotids is visible; and in aged persons the pulsations of the smaller superficial arteries may be generally distinguished by the eye [482]. (*g*) Præcordial pain, rare in simple hypertrophy, is not uncommon in the dilated variety, ranging in severity from a slight aching sensation to the severe suffering of pseudo-angina. Paroxysms of such pain may be accompanied with, and probably depend sometimes on, congestion of the lungs and loading of the right cavities of the heart with blood; in some instances they are distinctly traceable to intercostal neuralgia. The common action of an hypertrophous left ventricle would be palpitation, if not in frequency, in force, to a healthy person; under excitement, or often without apparent cause, a violent fit of throbbing action comes on—regular, however, or almost so, in rhythm, producing forcible pulsation in the neck and head, with tinnitus aurium. (*h*) I have not known hypertrophy produce albuminous impregnation of the urine, nor indeed any distinctive condition of the fluid. (*i*) It has been said the sexual propensity undergoes increase,—a statement I can neither affirm nor deny. (*k*) Cephalalgia, dull, aching, or throbbing, is of more frequent occurrence than in healthy persons, but by no means a constant symptom; sensations of rushing of blood to the head are common, especially on stooping, and, indeed, on sudden movement of any kind. The intellect is habitually unaffected, as regards any symptomatic state clinically significant; no proof exists of its being brightened; nor, on the other hand, unless towards the close of life, have I found pure hypertrophy render individuals incapable of ordinary mental exertion. (*l*) Reflex phenomena, at the moment the patient drops off to sleep, sudden starting of the legs, for example, are not very uncommon; they may possibly be traceable to active congestion of the spinal chord. (*m*) The eyes of some patients are bright, full, prominent, prone to injection; and by such persons visual illusions, luminous vision, and muscæ volitantes are frequently complained of. Epistaxis seems to be more usual than in individuals of equal age free from hypertrophy.

1307. Hypertrophy of the left ventricle is said to entail increase, in weight and substance, of the organs generally; and to enlarge the calibre, thicken the coats, and even increase the length of the arteries: but the age at which hypertrophy of the heart becomes common, is precisely that at which a natural tendency to enlargement of the vessels,—if indications given by the statistics of M. Bizot may be finally accepted,—is developed. The alleged influence of hypertrophy in producing renal disease with albuminuria, is not established: there may be some connection, through the fatty diathesis, between pseudo-hypertrophy or fatty enlargement of the heart and fatty alteration of the kidney; but I have seen nothing demonstrative of the connection.

1308. The influence of hypertrophy and other affections of the heart on organic changes in the brain is a matter of so much importance, that I may be excused for reproducing a passage of some years' date:—

> Nothing can appear more plausible than to suppose undue force of propulsion of the blood from the heart shall act as a cause of apoplexy, whether this be simply congestive (ictus sanguinis), hæmorrhagic, or dependent on red softening. Now, over-forcible propulsion of the blood through the systemic arteries is mainly the result of too energetic contraction of the left ventricle. Hence hypertrophy of the left ventricle has been, by a process of *à priori* reasoning, set down as a cause of apoplexy in general by some persons; by others, of cerebral hæmorrhage, in particular; by yet others, of red softening. Again, the doctrine referring white or colourless softening of the brain to deficient nutrition, occurring independently of inflammation, has had many followers. Persons taking this view of its nature are prepared to recognise, in any agency interfering with the distribution of a just amount of blood to the brain, an efficient cause of the disease. Now, constrictive disease of the aortic orifice, and regurgitant disease of the mitral orifice, must have this effect on the cerebral circulation, and so, it is inferred, must act as causes of cerebral softening.
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> It has been felt, I may almost say, on all sides, that these views require to be tested by facts; and the facts adduced, as far as they have reached me, may be summarily set forth as follows. First (*a*), in regard of "apoplexy," and "cerebral hæmorrhage," we find writers giving the subjoined numerical results of their observation concerning connexion between those cerebral affections and heart-diseases.

	Deaths from Cerebral Hæmorrhage.	Heart Hypertrophous.
Rochoux	42	3
Andral	17	9
D. Fardel	28	8
	Apoplexy with Hemiplegia.	Heart-Disease.
Clendinning	28	15
Hope	39	27
Burrows	34	23
	188	85

Calculating from the sums of these somewhat contradictory results, there seems presumptive evidence that the heart will be diseased in about 45·2 per 100 of cases of apoplectic seizure with sudden hemiplegia. But the precise ages of the persons supplying these figures is unknown. That it was advanced in the great majority of cases may be admitted from the known laws of cerebral hæmorrhage; that it was advanced in M. Fardel's patients is certain, for they were all observed at the Salpêtrière, where the rather patriarchal age of sixty years forms a condition of admission; the mean age in M. Andral's cases I calculate to have been 56·2 years. Now what proof have we that the number of hypertrophous hearts in the victims of cerebral hæmorrhage, just counted and percented, is greater than it would prove in a like number of aged people, cut off by all diseases, indiscriminately, affecting other organs than the brain? Absolutely none. So far as evidence goes, this seems, on the contrary, to show that the inference of close nexus of cause and effect, in cases where the cardiac and cerebral diseases were found, is unsound. Thus M. Fardel carefully examined the bodies of sixty aged persons (at the same institution, the Salpêtrière), cut off by other than cerebral causes, and found the heart sound in forty-five, hypertrophous in fifteen, of the number. In other words, we may expect that 25·0 per cent. of a mass of aged persons cut off by all diseases indiscriminately (except those of the brain), will have an hypertrophous heart. Now, this proportion of 25·0 per cent. is only twenty per cent. less than that of diseased hearts, furnished by the above series of apoplectic persons.

I have separated the results of the six authors quoted into two sub-series. The separation is an important one enough. The first sub-series refers especially to "cerebral hæmorrhage" in connexion with "hypertrophy;" in the second, the association is between "apoplexy" and "heart-disease," no precise affirmation being made, or intended, as to the condition productive of apoplexy, or as to the nature of the heart-affection. It is a striking fact that the first sub-series gives a proportion of hypertrophous hearts of only twenty-three per cent.—absolutely ten per cent. less than in aged persons wholly free from cerebral affection.

Let us next examine the question in the converse point of view, and see in what proportion of persons having hypertrophy, or other disease of the heart, as their main affection, cerebral apoplexy superveues. Here the following numbers are available for our purposes :—

	Disease of Heart.	Cerebral Apoplexy.
Ravier . . .	10	1
Louis*	45	0
Blakiston . . .	155	14
	210	15

So that 7½ per cent. only of persons labouring under diseased heart, for a greater or less number of years, became the victims of cerebral apoplexy.

Secondly (*b*), in regard of cerebral softening, the following figures may be adduced :—

	Brain softened.	Heart hypertrophied, or hypertrophied and dilated.	Aortic constriction.	Mitral regurgitation.
Andral .	33	9	3†	—
D. Fardel	41	8	—	2
Rostan .	18	12	—	—

From these figures it would follow that 35·9 per cent. of persons having a softened brain had hypertrophy, or hypertrophy and dilatation of the heart,—scarcely more than M. Fardel's average for aged persons dying of all diseases indiscriminately. In the series just given, Rostan's figures all refer to old persons, and those of Fardel to persons aged upwards of fifty. The latter observer affirms that he always found the heart healthy in patients cut off by softening before the age of fifty. The mean ages in Andral's cases of softening I have calculated as follow :—Where the heart was sound, 49·9 years; where there was hypertrophy, or hypertrophy and dilatation combined, 47·7 years; and where there was constrictive disease of the aortic orifice, 63·7 years.

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* I heard this numerical statement made by its author in 1837.

† One of these counts under the head of hypertrophy also.

Eight cases have been adduced by Dr. Law, to show that colourless softening of the brain (which he, with many other persons, holds to be allied in nature to gangrene of other textures) is at least frequently dependent on regurgitant disease of the mitral valve. Four post-mortem examinations only were made in these cases; the *green* tint of the softened parts justifies the suspicion that pus may have been present.

From this rapid survey of the subject of the connexion of cardiac and cerebral diseases, what inference can fairly be drawn? None other, I think, than that positive asseverations of the power of heart-disease to generate brain-affections, *as a demonstrated fact*, had best be avoided. On the other hand, I believe that it would be just as unsound to deny totally the existence of any such power on the faith of the numerical comparisons I have just instituted. These are the only comparisons of the sort obtainable at the present hour, but let us not shut our eyes to their serious imperfections. These imperfections are of different kinds. In the first place, some writers are so deeply prejudiced on the question at issue, that their facts cannot be received otherwise than with some quantum of distrust. Read the pages of Andral, and observe how determined he is to find the nexus of an hypertrophied left ventricle, and cerebral hæmorrhage; read those of Rochoux, and note the eager partisanship with which he strives to disprove it. Do this, and I feel satisfied you will, with me, see the wisdom of receiving *cum grano salis* their general conclusions. In the second place, some writers put forward cases where no post-mortem examination took place; these are, under the circumstances, unfit to form elements in the discussion. Thirdly, who can for a moment suppose it to be at all likely, that all varieties of heart disease shall have an equal tendency to produce cerebral congestion? That, for example, an hypertrophous left ventricle shall do the same violence to the minute cerebral vessels, if it play through a constricted aortic orifice, as if it play through a perfectly free one. Who can suppose that where the radial pulse is small, feeble, unequal, tremulous from highly developed insufficiency of the mitral valve, the blood shall be propelled with excess of force into the small arteries of the brain? Far from this, the contrary appears so fair an hypothesis, that, as we have just seen, an experienced physician regards such insufficiency as an efficient cause of gangrene-like softening of the brain. The whole clinical history of mitral regurgitant disease points to pulmonary, and not to systematic congestion. And, *per contra*, the ascertained effects of tricuspid regurgitation give an air of probability to the view that this variety of cardiac imperfection may in reality form the true link between congestive affection of the brain and the heart. Whether this be the fact or not, time will show. Meanwhile, I think there can be no question that it is deeply unsound to club together cases of such opposite functional tendencies as those we have been noticing, and regard them as a single mass producing one single definite effect on the brain. Fourthly, an objection to some of the cases figuring in the returns I have given you lies in the fact, that the respective dates of the cerebral and cardiac diseases have not been clearly made out. And again, fifthly, the subject would require revision, were it only for the change recently effected in our knowledge of the morbid anatomy of enlarged hearts. All enlarged hearts were formerly set down as enlarged by muscular hypertrophy; we now know that the available muscular substance may be less than natural in such hearts, encroached upon and impoverished as it is by accumulating fat. And let us not appeal here to clinical experience to set aside the inference deducible from post-mortem investigation. True, the action, felt in the præcordial region, of a fat-infiltrated heart may be agitated and forcible, but violence of *impulsion* does not signify power of *propulsion.*

The correctness of the views here taken seems clearly supported in one aspect by the recent observations of Dr. Kirkes on the detachment of fibrinous coagula from the left side of the heart.

1309. Drs. Parry, Graves and Stokes, direct attention to enlargement of the thyroid gland and inferior thyroid arteries as a dependence on permanent excitement of the heart's action. Dr. Stokes states that, in fatal cases, though the cardiac disturbance be originally dynamic, dilated hypertrophy is found,—that, more common in the hysterical female, thyroid enlargement still does occur in the male,—that the enlarged gland pulsates and furnishes the signs of aneurismal varix, all of which signs may disappear with increasing solidity of its structure.

1310. *Prognosis.*—Simple hypertrophy, of medium amount, may be rendered a very endurable affection by means of regulated diet, moderation in exercise, and general attention to hygienic rules. If the disease be carried to a high point, if it be of considerable amount, and the patient, instead of living according to rule, is forced to work laboriously, and live irregularly, the probable issue is death, through secondary affections, complications and functional derangements, which, though originally unconnected with the heart-disease, are rendered more serious by its existence. But hypertrophy alone is rarely, and I do not think ever rapidly, the direct cause of death: I cannot call to mind any case, where I have actually known it, and it alone, positively fatal. Dr. Hope, it is true, affirms he has known hypertrophy destroy life in some instances within a year of its commencement (Op. cit., p. 278); still he cites no cases, and appears from the context to have in his mind's eye examples of dilated hypertrophy variously and seriously complicated. Dr. Latham is of opinion, "the heart, *by the simple vehemence of its action*, has the power to kill,"—through cephalalgia, insomnia, delirium, mania, convulsion, and nervous exhaustion (Diseases of the Heart, vol. ii., p. 338). But by the phrase, "simple vehemence of action," we are not to understand, however this may appear warranted by its terms, such action as an unaided hypertrophous heart in its highest degree can engender. . For Dr. Latham gives no proof that the organ possesses any such power, and the only positive case referred to in illustration of the above opinion, is one of hypertrophy and dilatation of the left ventricle following endo-pericarditis and bygone dropsies;—where, too, as no *post-mortem* examination took place, there *may* have been disease within the cranium.

The more dilatation predominates over hypertrophy, the more serious becomes the prognosis.

1311. With what hopes may the *treatment* of pure, or somewhat dilated, hypertrophy of the left ventricle be undertaken? Is the disease curable? Can the nourishment of the heart be not only controlled by artificial means, but reduced below the standard of health? Drs. Latham, Taylor, and Blakiston emphatically deny that art is possessed of any such power; Laennec and Dr. Hope maintain that the feat is easy of accomplishment. But Dr. Hope repudiates the plan recommended by Laennec, and the simple common-sense system lauded by himself fails utterly in effecting the textural changes he ascribes to it. For my own part, I have never known the cure of indubitable hypertrophy proved by physical signs, and hold it unwise to promise any such result from treatment.* But it is not difficult to remove or greatly mitigate the symptoms of simple hypertrophy in the majority of cases, and render life not merely tolerable, but comfortable.

1312. The theoretical indication is very obviously to tranquillise the heart by diminishing the quantity, without deteriorating the quality, at least materially, of the circulating fluid. For this purpose, occasional bleedings from the arm, to the extent of four, six, or eight ounces at a time, at intervals of from two to six weeks, are recommended by Dr. Hope, in conjunction with a diet mainly consisting of farinacea and vegetables. But even loss of blood to the extent here intended, is more than can be borne by the majority of persons, without slight impoverishment of the fluid ; and the least amount of anæmia deeply aggravates the dangers of hypertrophy. Besides, it has appeared to me that over-action of the heart is quite as effectually and as lastingly controlled by very moderate cupping or leeching over the præcordial region, as by the abstraction of a comparatively large quantity of blood from the arm. Four or five leeches, even, will sometimes calm the excitement of a powerful left ventricle, in a state even of somewhat dilated hypertrophy,—and this in a well-grown adult.

1313. There is no known drug possessing the faculty of directly controlling the growth of the heart ; iodine seems quite valueless : general principles alone guide us in attempting to put a term to the increase of its bulk. Purgative medicines, and I believe the saline and aloetic the most appropriate of the class, aid the good effects of local bleeding. Diuretics are useful, quite independently of the existence of dropsy. Direct sedatives of the heart, hydrocyanic acid, acetate of lead, digitalis, and belladonna (the latter both internally, and in the form of plaster to the surface) must be employed, with occasional intermissions, during the entire treatment of the case. Of all medicines of this class aconite, however, seems the best; the alcoholic extract of the root may be given in doses of one eighth of a grain with perfect safety in cases of this species; no drug, that I know of, possesses so fully the power of relieving painful sensations and disquietude about the heart.

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* Even the physical signs may, unless he be very cautious, betray the observer into error in regard of this matter. Thus, not only the impulse may be reduced in force and extent, and the character of the first sound changed, but the area of percussion-dulness lessened, by disgorgement of the right cavities, and yet the heart's actual mass remain precisely as before. All this may sometimes be done, by treatment, in a few days ; sometimes not in months.

1314. Unless the patient be very highly plethoric, animal food in moderation should be allowed,—under all circumstances, indeed, fish may be permitted. Alcoholic fluids, of all kinds, must be avoided; and liquid taken, as a rule, in but small quantity. Moderate exercise is advisable.

1315. In treating a case of this kind, the patience of the physician must never fail him: it may require months, nay, years, to produce a favourable effect on the disease,—and want of steadiness of purpose and conviction may, in a few days, undo the good accomplished by the efforts of previous weeks. There is one caution to be given to the young practitioner,—that he never push the treatment to the extent of producing anæmia; the super-addition of anæmia to hypertrophy is that which gives a really ominous character to the latter.

1316. II. *Concentric Hypertrophy of the Left Ventricle.*—The discovery that the contraction of the ventricle occurring *in articulo mortis* might give to a heart simply hypertrophous the appearance of one concentrically hypertrophous, led, for a time, to the almost complete rejection of concentric hypertrophy as a possible state. But its occasional, though rare, existence is matter of asserted fact. The symptoms and signs are those of simple hypertrophy; theoretically the disturbance of the circulation will be greater, but clinical illustrations of the point are wanting. It has not occurred to me to meet with a pure case of the kind.

1317. III. *Hypertrophy of the Right Ventricle* is comparatively rare in all forms, and practically unknown, unless associated with dilatation.

1318. The signs of its dilated hypertrophy are arching of the lower part of the sternum, with greater or less eversion of the ensiform cartilage, and fulness of the epigastrium. The left costal cartilages may be more bulged than the right,—a circumstance accordant with the fact, that though the impulse plays very forcibly against the sternum and ensiform cartilage, it inclines, in cases of highly marked right hypertrophy, to direct itself, in consequence of slight displacement of the entire organ, more against the edge of the left, than the right mammary region. Hence it is, that the form of the præcordial region and the site of impulse might betray the observer into the notion that the left, and not the right, ventricle was the seat of hypertrophy. The percussion-dulness may extend considerably beyond the right edge of the sternum; I have known it reach an inch outside that edge in a child aged ten years: at the same time the dulness may be carried unduly to the left also. The distended and enlarged right auricle is the source of much of the percussion-dulness to the right of the sternum.

Dilated hypertrophy of the right ventricle widens the tricuspid orifice in the majority of cases; unless the valve grow in proportion to the widening of the orifice, a regurgitant murmur should, theoretically, occur. Yet, such murmur certainly does not occur by any means constantly, in cases where, *post mortem*, the valve appears physically incompetent. Can this be explained by some constrictive action of the orifice during life preventing regurgitation [507]? The first sound is duller than natural at the ensiform cartilage; the second fuller, stronger, and more accentuated. Visible jugular pulsation sometimes exists, without the tricuspid valve being demonstrably incompetent.

1319. Marked hypertrophy of the right, is so rare without hypertrophy of the left, ventricle, or some form of valvular disease, that it is difficult to give a really clinical transcript of its symptoms. Lividity of the face, and subcutaneous œdema about the face and neck sometimes exist. Theoretically the lungs must suffer,—and hence dyspnœa, engorgement and œdema of the pulmonary parenchyma, bronchitis, pneumonia, tubercles, pulmonary apoplexy, and hæmoptysis, have all been set down by various systematic writers, as dependencies of the affection. Now, of all these alleged symptoms, dyspnœa is the only condition that seems to have thoroughly made out its claim to be so entitled. I believe, however, that engorgement and œdema of the tissue of the lung is not uncommon. Pneumonia, pulmonary apoplexy, and hæmoptysis, I have certainly seen in cases of right ventricular hypertrophy; but in these instances, mitral regurgitation existed also. The notion that right hypertrophy tends to tuberculise the lungs, is not supported by anything I have observed.

The radial pulse, from its being free from all peculiarity of character, affords valuable aid in the diagnosis. Natural in force, while the cardiac action is strong, its state argues against the existence of hypertrophy of the left ventricle, to which some of the local physical signs might otherwise point. The pulmonary artery, of course, bears the force of the hypertrophous ventricle; a fact probably explanatory of the frequency with which, under these circumstances, its coats are atheromatous.

The difficulty of circulation through the vena cava and jugulars tends to congest the brain venously.

1320. *Hypertrophy of the Auricles.*—Hypertrophy of the left auricle often accompanies, in variable degrees, constrictive and regurgitant disease of the mitral orifice: dilated hypertrophy of the right auricle, with chronic thickening of its endocardium, I have seen carried to a great extent in cases of dilatation of the tricuspid orifice. The symptoms of the diseased state of orifice, whatever they are, are intensified by these auricular hypertrophies; of signs peculiar to them, there are few [362, 427].

CARDIAC DROPSY AND FLUX.

1321. I. *Dropsy.*—Effusion of the serum of the blood into certain parenchymata and membranous sacs is one of the most important symptoms connected with various affections of the heart,—and, as its precise relationship to individual cardiac diseases must still be considered *sub judice,* the subject may, with propriety, be separately examined.

1322. Cardiac dropsies are preceded immediately by venous congestion, itself produced by conditions to be by-and-by inquired into. The fluid composing them, of low specific gravity, contains but a small proportion of albumen, does not coagulate spontaneously, is free from urea, and, as far as I know, any other excrementitious principle, and never forms a blastema apt for evolution.

1323. Dropsy, depending on heart-disease, occurs in the following situations, enumerated in the order of frequency with which they severally suffer: the subcutaneous cellular tissue; the pulmonary parenchyma; the peritonæal and pleural sacs; the pericardium; the cerebral and spinal arachnoid, and sub-arachnoid, spaces; the tunica-vaginalis; the joints; and the eye-ball, especially the aqueous chambers. But in the last three situations dropsy is excessively rare.

1324. Cardiac anasarca commences almost invariably about the ankles and feet, gradually extending upwards: still I have known it originate about the eyelids, where the kidneys were functionally and texturally healthy; such cases, excessively rare, belong to the class of dilatations with tricuspid regurgitation. When carried to great amount, erythema, erysipelas, and even sloughing are prone to occur: the skin cracks, and through the fissures a fluid, at first glutinous, afterwards watery, oozes more or less copiously.

1325. Dilatation was formerly regarded as the condition of the heart mainly inducing the important class of dropsical symptoms. This opinion, discountenanced by Bouillaud, under the apprehension that valvular obstruction was their sole cause, was restored to favour by Hope and Andral,—the former of whom, indeed, went the length of teaching that pure hypertrophy, also, was capable of generating dropsy. Of late, Dr. Blakiston has brought together a body of evidence calculated to show that the systemic vessels do not become loaded in cases of dilatation, unless there be co-existent tricuspid regurgitation. Still more recently Dr. H. Douglas has defended the original thesis of our forefathers. It becomes necessary for me here to express an opinion on this "vexed question;" and I will do this by throwing into a series of propositions such inferences as flow directly from facts that have fallen under my own notice.

1326. 1. Mitral regurgitation or obstruction, or aortic regurgitation or obstruction, may severally exist, and, for a lengthened period, without systemic dropsy supervening. 2. Mitral regurgitation and aortic regurgitation may co-exist for years, and yet no dropsy occur. 3. Both of these propositions hold good, whether notable hypertrophy do, or do not, exist behind the obstruction. 4. Simple hypertrophy of the left ventricle may reach the highest point without systemic congestive effects of any kind arising. Dr. Hope, as is well known, maintained the reverse, holding that pure hypertrophy, if protracted, will produce general dropsy; but he gives no cases demonstrating the fact, and the motives of his belief are, as far as he shows, totally speculative. And *à priori* views are not so completely in his favour as he appears to imagine; when he talks of the "increased force of circulation surmounting the natural tonic power of the capillaries," he forgets that that very tonic power may have increased *pari passu* with, and in consequence of, the growth of the hypertrophy. The question is one of observation; and I have stated what I have actually seen in persons who had not been reduced by treatment to a state of anæmia. 5. Dilated hypertrophy, even, of the left ventricle, may last for years without any such effect, provided the dilatation be not in notable excess. 6. The heart may be weak and feeble, or actually in a state of fatty disorganisation, and the pulse feeble and irregular, and yet no systemic congestions occur. 7. The natural relationship of width of the arterial orifices, and also of the auriculo-ventricular orifices, may be materially perverted, without the least systemic dropsy arising, until the closing days of life.* 8. Tricuspid regurgitation, where the right ventricle is in a state of dilated hypertrophy, as shown during life, by swollen and pulsatile jugular veins which fill from below, and as shown after death by actual examination, does not necessarily produce dropsy.† 9. It would appear, then, that something beyond all these cardiac conditions is wanting to ensure the occurrence of dropsy, unless on the gratuitous assumption that, were life sufficiently prolonged, they would in themselves of necessity induce it. 10. The existence of some active cause beyond, and independent of, the heart, is further shown by the facts: that there is no direct relationship between the amount of heart-disease and of dropsy; that dropsy comes on suddenly sometimes from extraneous causes, the state of the heart remaining, as far as ascertainable, in precisely its previous condition; and that dropsy diminishes and increases, comes and goes, either spontaneously or through the influence of treatment, while the organic changes in the heart remain permanent and unmodified. 11. The cardiac affections most frequently connected, as matter of experience, with systemic dropsy, are dilatation and tricuspid regurgitation; and it is certainly very rare for either of these states to exist for any length of time without the supervention of such dropsy: any hypothesis, explanatory of cardiac dropsy, must look to these states as forming important links in its chain of causes.

1327. The question next arises, what is the nature of the influence, independent of the heart, which leads to dropsical effusions? This influence seems a compound of conditions, favourable to transudation of the serosity of the blood, in that fluid itself, in the walls of the capillaries and venous radicles, and in the receiving tissues. First, as concerns the blood, the influence of an impoverished state of that fluid is too well known to be for a moment contestable. Experiments on animals prove that if the veins be more or less loaded with water, they yield this in the form of dropsical effusion. The œdema, and sometimes extensive anasarca, of spanæmia; the slight œdema attending the hypinosis of protracted convalescence from various acute diseases; the various dropsies of uræmia,—are all illustrations in point. Obviously morbid states of the blood, when of the proper kind, of themselves alone suffice for the production of dropsy: look at the sudden anasarca of acute Bright's disease, or of an acute recrudescence in the chronic affection, while the heart, liver, and lungs, may be texturally sound. Secondly, it is readily conceivable that the variable density of texture of the walls of the vessels shall promote or restrain the process of filtration. Thirdly, cases occasionally present themselves, in which dropsy, supervening from diseased heart, fails to affect portions of the body, noted, under ordinary circumstances, as the earliest and readiest sufferers,—for instance, the lower extremities. I have observed this where the legs had been the seat of erysipelas and subcutaneous inflammation prior to the occurrence of the cardiac dropsy: the chronic anatomical changes in the cellular tissue in such a case possibly act as a barrier to its reception of serosity from the vessels.* Dilatation of the heart, occurring as a primitive disease through simple weakness, or actual structural alteration of the texture of the organ, may be easily supposed *à priori* capable of generating systemic congestion and its results. In a heart so affected, the necessary *vis à tergo* is deficient,—capillary stagnation ensues. Now this very stagnation, becoming habitual, may modify the qualities of the blood; and impair the nutrition of the walls of the vessels through the strain they suffer. But change in the blood is, in all probability, worked out meanwhile by other and more effectual agencies.

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* Case of Cyanosis, Med. Chir. Trans., vol. xxv., p. 1.

† W. Hallington, ætat. 53, died at U. C. H., April 7, 1849, with fatty dilated enlargement of the heart (weight 17½ oz.), dilatation of the tricuspid and mitral orifices, and aortic regurgitation carried to a great extent, and yet not a particle even of œdema about the ankles had appeared. The patient had also an aneurism of the aorta, at its bifurcation; but this could not be held to be *preventive* of dropsy.

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* Clin. Lect., case of Hope, "Lancet," loc. cit., p. 442. Robertson, U. C. H., Females, vol. viii., p. 295.

Long since, Lower showed that local dropsy might be produced by ligature of veins; and the occurrence of serous effusion from local obstruction of vessels of that class is clinically well known. But here are instances of sudden, and limited obstruction; they are evidently not logically comparable with cases of slow and centric obstruction.

1328. The *treatment* of cardiac systemic dropsy must vary somewhat with the precise conditions of the heart; but there are some points of general applicability which may here be set down. There is no period of valvular disease at which the removal of dropsy may not be accomplished; but when the powers of the patient are seriously enfeebled, great caution, as regards the rapidity of that removal, is called for.

The dropsies of heart-disease are very rarely attended with true plethora, and hence on that score very rarely call for venesection. But, when a clogged condition of the heart's cavities coexists with pulmonary congestion and anasarca of more or less acute course, and the general vigour is as yet unimpaired to any serious degree, the abstraction of a small quantity of blood, either by venesection or cupping of the chest, has appeared to me to facilitate the action of hydragogue remedies. That diminished fulness of the vascular system promotes absorptive power, is well known; but, even where the quantity of blood abstracted scarcely authorises us in resorting to this explanation, the result is beneficial.

Of hydragogue purgatives, peculiarly valuable from their certainty of action, elaterium,* gamboge, bitartrate of potass, and the pulvis jalapæ compositus are the most valuable of the class. Stimulants may be requisite during the action of these medicines; and their action may be so rapid, that the mere loss of fluid shall weaken vitally and mechanically. An ounce and a half of the bitartrate of potass may be given with perfect safety, freely diluted and combined with carminative tinctures,—smaller doses having first been employed.

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* The formula I employ is as follows:—R Extract. Elaterii. gr. ⅛—gr. ¼; Creasotonis, gt. i.; Extract. Hyoscy., gr. ii.; pro pill., i. The inequality of

Diuretics, uncertain in their action, are often rendered much more active by preliminary cupping or dry-cupping of the renal regions. It will be necessary to vary the combination from time to time, and the list for selection is fortunately a full one,—comprising the acetate, nitrate and bitartrate of potass, the iodide and bromide of potassium,* nitric ether, the spirits and infusion of juniper, squill, colchicum, decoction of scoparium and of chimaphila, and, if the heart's strength be not seriously impaired, infusion of digitalis, or if the liver be at all engorged, the extract and decoction of taraxacum. Hydragogue purgatives often promote the action of more direct diuretics; and small doses of blue pill, occasionally at bed-time, are very useful adjuvants.

Diaphoretic drugs are of little utility; but the free diaphoresis produced by the vapour, or hot-air bath, is sometimes strikingly beneficial.

If there be any tendency to bronchitis, bronchial discharge should be encouraged by the free use of expectorants.

Removal of anasarcous fluid by mechanical means becomes necessary, if medicines fail in controlling its increase; and if inflammatory changes in the skin appear imminent. Scarifica-

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strength of the extract, as supplied by various druggists, renders it prudent to commence with a very small dose: the greater certainty of the strength of the alkaloid, will probably lead one day to its being generally employed, though with a purgative of such intense activity, and at the same time, occasionally depressing character, the greatest caution must, of course, be observed.

* The iodo-bromuretted waters of Adelheidsquelle, Kreuznach, and Woodhall, act diuretically in some cases.

tion is hazardous,—being not unoften followed by erysipelas; while acupuncturation at the upper and inner parts of the thighs, though a slower process, is not attended with the same danger: there is even some advantage in the slowness of discharge; too rapid evacuation has been followed by great depression. On the other hand, should discharge have commenced, either through spontaneous fissures or artificial punctures, it is extremely dangerous to arrest the flow by healing the openings: I have known death rapidly ensue from dyspnœa and thoracic dropsy under the circumstances.

The patient's strength must be supported by food containing much nutriment in a small compass; and, though thirst torment him, he must refrain from fluids as far as possible. Stimulants may be required; and hollands or gin have both popular prejudice and medical experience in their favour under the circumstances.

1329. *Pulmonary œdema* is chiefly connected with disease of the mitral orifice,—sometimes with dilatation of the left ventricle, rarely with hypertrophy of the right. *Ascites* arises especially from general dilatation and tricuspid regurgitation; and these are the conditions mainly observed as antecedents to all the other varieties of dropsy enumerated.

1330. II. *Flux.*—Intestinal serous flux, and hydruria, occasionally occur spontaneously in the course of cardiac disease: they probably ward off dropsy.

DILATATION OF THE HEART.

1331. By dilatation of the heart, is understood that state in which the capacity of its cavities is increased disproportionately to the thickness of their walls. It occurs in three forms: *hypertrophous dilatation*, where dilatation predominates, but the walls are somewhat thicker than in health; *simple dilatation*, where the walls are of such thickness as would be normal, had the capacity remained unchanged; and *attenuated dilatation*, in which the walls are distinctly thinner than in health,—to such a degree that the thickness of the wall of the left ventricle may, in some points, positively not exceed one line. Clinically, as well as anatomically, the characters of dilatation are obvious in proportion to the excess of capacity over thickness; and the following description must be understood to refer to cases in which such excess is prominently marked. Dilatation of this kind most frequently affects both ventricles; one, however, to a greater amount than the other: it is not uncommonly limited to the right, very rarely to the left, ventricle. The form of the dilated ventricle becomes globular, and the apex of the heart so rounded off, that it may with difficulty be detected. The transverse measurement of the organ undergoes much greater increase than the vertical.

1332. Dilatation of the orifices only occurs, as a rule, where both auricles and ventricles are affected on the same side: insufficiency of either auriculo-ventricular valve may thus be produced independently of disease in itself. There is a tendency to accumulation of blood in the cavities; at least grumous coagula are commonly found,—the tendency being sometimes, though rarely, counteracted by the mode of death.

1333. The tissue of the dilated walls is more or less soft and flabby—and if this deficiency of firmness be disproportionately marked at any particular spot, the ventricle may there yield, undergo pouching, and a true aneurism of the heart ensue. The colour may be deep brownish red, anæmically pale, or mottled yellowish brown. Microscopically, the fibre is deficient in striated character, and granular looking; fat, extra- or intra-sarcolemmous, may exist; and in some cases the appearances of adventitious cellulo-fibroid texture are traceable, the muscular substance being atrophous.

1334. *Physical signs.*—The physical signs of general dilatation may be set down somewhat as follows:—The apex-beat is very indistinctly visible, or, if the patient be full in person, actually invisible; when seen, it may fall *within* the natural point of the apex-beat, in consequence of the rounded-off form of the heart destroying, or lessening the clearness of definition of, the true apex; if the dilatation be considerable, the apex strikes the surface *below* the fifth interspace. The impulse, whether more extended than natural, or limited to the fourth and fifth interspaces, frequently exhibits the quasi-undulatory character. There is no prominence of the cardiac region. As felt by the hand, the shock is feeble in proportion to the purity of the dilatation: the apex-beat, though visible, frequently cannot be distinguished by the finger from the impulse of the ventricles generally. The impulse, non-vibratile, either consists of a short feeble slap, followed by a sudden fall back of the organ, or of a more prolonged faint tremulous motion: the force of successive beats is unequal; their rhythm may be irregular to a slight or to the very highest degree: there is a want of perfect uniformity in the point of the surface struck by the heart in the successive beats of a series, quite independently of the influence of respiration: palpitation may notably increase the force of impulse, though it often does so but very slightly. The intensity of percussion-dulness in the superficial cardiac region is not, as it probably is in hypertrophy, increased; and in cases of attenuated dilatation the parietal resistance may be less marked even than in the state of health. The areas of dulness, deep and superficial, are both widened; the former inclines to squareness of outline, the transverse diameter of the heart having proportionally undergone a notably greater increase than the vertical. The systolic sound, short and abrupt, may be unnaturally clear, both at the apex and base, if the heart's tissue be firm; it appears to be produced near the surface, and its maximum point is generally slightly lowered. The diastolic successor is not specially affected. Both sounds, as heard at the top of the sternum, bear the natural relationship, in point of intensity and clearness, to those audible over the heart itself. If the dilated ventricle be soft, flabby, or fatty, the first sound may be very weak, faint, and toneless; and the second so feeble as to be inaudible at the apex, especially if the heart's rhythm be markedly irregular. Upon the quality and mass of the heart's texture will depend the extent to which the sounds are transmitted through and over the chest; if the dilated ventricles be well nourished, and *à fortiori* at all hypertrophous, the sounds (approaching those of hypertrophy with dilatation) will be widely diffused, and clearly audible in the right back; if, on the contrary, the dilated heart be a soft, flabby one, the area over which its sounds are audible may be extremely limited. A slapping noise, the intensity of which may be increased by causing the patient to bend forwards, is sometimes produced by the collision of the heart against the side. Dilatation is not habitually productive of any intra-cardiac murmur, unless the morbid state has proceeded to such lengths as to render certain valves incompetent to close their orifices: this may occur with the tricuspid, more rarely with the mitral, and still more rarely with the aortic valves. The murmurs thus engendered are, of course, always regurgitant; but they do not always occur when their physical cause, incompetency of the valves, actually exists, inasmuch as the strength of the ventricles is sometimes incapable either of giving the necessary impetus to the regurgitating stream, or of calling forth such energy of its own systolic reaction on the part of the aorta, as is required for the production of murmur. It is also possible that dilatation, by giving an unnatural direction to the blood-current, may engender murmur [436, 442]. Are reduplications common when the ventricles are unequally dilated? I think so; but my number of observations on the point is too small to justify a positive assertion.

1335. Dilatation of the right ventricle in particular is signified by extension of percussion-dulness to the right, by excess of epigastric pulsation, by jugular turgescence, and, if there be either tricuspid regurgitation or some muscular strength in the wall of the ventricle, by jugular pulsation.

1336. Direct evidence of dilatation of either auricle is only to be had by percussion in the natural sites of those cavities: when the jugular veins are permanently dilated and knotty, the existence of dilatation of the right auricle is inferrible almost as matter of necessity.

1337. *Symptoms.*—Taking, as our imaginary model, a case of highly marked general dilatation of the heart, the failures of the various functions will be found much of the following kind:—(*a.*) The patient, habitually irritable and melancholy, breaks from time to time into fits of despondency and petulant complaint; he is deficient in energy, both mental and bodily—the dislike to movement is only overcome under the persuasion that exercise is in some measure essential; the strength fails; the weight, unless factitiously sustained by dropsies, falls very notably; sleep, in the true sense of the term, is rarely enjoyed,—fitful dozes, abruptly interrupted by startings and frightful dreams (incubus is not more common than in persons free from heart-disease), are its nearest substitute; habitually the patient lies with his head high, and when under the pressure of a fit of dyspnœa, or cardiac asthma, he bends the head forwards, or sits erect with the feet hanging out of bed; his debility and dropsical unwieldiness prevent him from assuming some of the peculiar postures in which the subject of pulmonary spasmodic asthma struggles for breath. But there are cases of well marked dilatation in which the patient habitually keeps the head low; I have known it kept as low as the shoulders: the cause of the peculiarity is far from clear.* (*b.*) Chilliness of the extremities, livid discoloration of the prominent parts of the face, mottled with patches of sallow and earthy tint, or varied by leaden or almost black discoloration about the eyes and mouth,—conditions of colour all of them most marked, as a rule, in the morning: lividity or blueness of the lower extremities generally, with excess in particular spots,—spots which, eventually falling into a state of absolutely stagnating circulation, slough, independently of calcification or inflammation of the arteries or veins;

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* A patient with dilated heart, cardiac dyspnœa, congested lungs, and hydrothorax, may be easiest with the head low. Mr. L. seen with Mr. Hargraves, Tunbridge Wells, Sept. 1852.

and, lastly, anasarca, softly pitting, spreading from the feet to the abdominal, and even the thoracic, walls, the external genital organs, the face and neck, rarely the upper extremities; all these conditions show how deeply the tegumentary system suffers. (*c.*) The joints remain unaffected. (*d.*) Anorexia and nausea may be habitual. The tongue is broad, full, not always pitted at the edges, however, and of dark purplish ground,—the fauces venously injected. Hæmorrhage may occur from various parts of the alimentary mucous tract; I have seen this in the case of the bowels, when the dilatation alone seemed its direct cause, but do not feel positive that hæmorrhage from the stomach has occurred under my observation, unless there were intermediate high congestion of the liver,—the hepatic system may act as a sort of safety-valve for the stomach, until itself grows overloaded. Hæmorrhoids are not so common as might be expected,—the bowels habitually constipated, or alternately relaxed and confined,—the discharges dark. The liver is more or less congested: the hepatic system suffers first, the portal secondly; unless both are implicated, the size of the organ is not sufficiently increased to affect the results of percussion notably: the texture of the lobules may remain perfectly sound. Positive jaundice does not occur from this state of the liver alone; but I have seen a faint yellowish tint during life, where death disclosed nothing but hepatic congestion to account for it. Ascites follows on (I have no experience of it as a precursor of) anasarca. (*e.*) Venous stagnation, with its consequences, occurs in the lungs,—oppressed or sighing breathing, eventually orthopnœa, complete and habitual, with occasional asthmatic paroxysms, and cough, dry, harassing, and even convulsive, or accompanied with expectoration, serous, rarely frothy, or stained or streaked with blood, or mixed with a little blood, fluid and dark, or in pellets. The physical signs of bronchitis, or of pulmonary congestion irregular, though most marked at the posterior bases, of pulmonary œdema, or of pulmonary apoplexy, may be found,—actual hæmoptysis to some amount may accompany the latter state. (*f.*) Feeble, fluttering distressing palpitation, increased by the slightest movement, occurring from some obvious cause, or as frequently without apparent excitement, is a standing source of misery to those sufferers. So, too, is uneasiness in the cardiac region, of characters most difficult to describe,—of an intensity varying between a mere sensation which constantly reminds patients that, as they often express themselves, "they have a heart," and the agony of angina,—paroxysms of which may actually occur and put an end to existence. Tenderness on pressure may be discovered over the heart in some cases. The pulse is either small and feeble, and abnormally posterior in time to the ventricular systole, but regular; or it is narrow, feeble, fluttering, and irregular. The latter state is either limited to the periods of palpitation, or, if constant, is indicative generally of softness of the heart's texture. True intermittence is rare. The superficial pulses are not visible. Faintness, occasionally lapsing into actual syncope, occurs from time to time. (*g.*) Swelling of the abdominal lymphatic glands is said sometimes to take place: I have never seen it. (*h.*) The kidneys are passively and mechanically congested like the lungs and liver; the renal regions may be tender on deep pressure posteriorly; but I have never known the organs sufficiently increased in bulk to cause any positive extension of the area of their posterior lumbar dulness. The urine is small in quantity, high coloured, loaded or not with lithates; albuminuria may occur from mere renal congestion,—* the urine continues of good specific gravity, and exhales no whey-like odour. The symptoms often disappear spontaneously, and may almost always be quickly removed by certain measures directed to the kidneys, especially cupping or dry-cupping. In rare instances spontaneous hydruria occurs temporarily. (*i.*) Sexual inclination, though weakened materially, is not annulled, — rupture of a dilated heart has occurred *in actu coitûs.* It is said that uterine hæmorrhage is sometimes traceable solely to dilatation of the heart: the statement, unconfirmed by any cases that have fallen under my notice, seems to stand in need of corroboration. (*k*.) More or less congestion of the encephalon can scarcely be escaped, where the face exhibits marked indications of that state. The dull cephalalgia, inability to exercise the mind, heaviness, torpor, somnolence, semi-coma, and finally, coma, that mark the progress of these cases, are thus explicable. Cerebral hæmorrhage and acute softening stand in undetermined relationship to dilatation. (*l*.) Congestion of the cord may be the cause of certain reflex phenomena, spasmodic cough, startings from slumber with affright, and convulsive actions of short duration on waking from sleep. (*m*.) Muscæ volitantes, luminous vision, fulness, wateriness, and injection of the eyeballs; tinnitus aurium, dulness of hearing, and deficiency of smell, and occasional epistaxis, from congestion of the Schneiderian membrane; all indicate participation on the part of the organs of sense.

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* Couch, U. C. H., Females, vol. vii., pp. 341—345.

1338. Taking the systemic and the pulmonary obstructions as two separate classes, the former have occurred under my observation almost invariably before the latter: Dr. Hope taught the converse, but, it appears to me, on theoretical grounds.

1339. It is difficult to affix specially to dilatation of the *right ventricle* any symptoms that may peculiarly depend upon it,—so frequently does some amount of dilatation of the left ventricle or some form of valvular impediment exist to complicate the problem. Theoretically, the signs of deficient oxygenation of the blood will be marked in proportion to the amount of pure or attenuated dilatation of this ventricle,—as will, also, the congestive influences on the brain and abdominal viscera.

1340. The *diagnosis* of dilatation of the heart turns mainly on the following points:—Weak action, quasi-undulatory impulse, indistinctness of the apex-beat; increased area, with squareness of outline, of the percussion-dulness, the whole not lowered in proportion to its width; clearness, shortness, abruptness of the systolic sound, or great deficiency of tone in certain cases, with prolongation of the post-systolic silence; peculiar characters of the pulse; and signs and symptoms of systemic and pulmonary obstruction and congestion.

1341. *Simple hypertrophy* is distinguished from dilatation by the forcible action of the heart; by the præcordial bulging; by its distinctly localised thrusting impulse; by the heart being lowered in proportion to the general area of dulness; by the muffled dulness and prolongation of the first sound and shortness of the first silence; by the characters of the pulse; and by the systemic signs being those rather of an excited than of a stagnating circulation. Every one of these characters differs more or less from those of dilatation.—*Eccentric hypertrophy*, also, has its forcible action, præcordial arching, and distinct apex-beat; the heart is lowered in the proportion of its bulk; the systolic sound is loud and powerful; and the pulse differs from that of dilatation. The only difficulty comes of the fact that if the cavities be more increased in capacity than the walls in thickness, the systemic and pulmonary signs may incline to those of pure dilatation.—*Mitral regurgitant* disease will, under ordinary circumstances, be distinguished from dilatation by its special systolic murmur at the left apex, and by the more forcible impulse: for some amount of hypertrophy in the vast majority of cases ensues on the valvular disease. Still there are two kinds of possible fallacy: dilatation may be attended with dynamic mitral murmurs; and, on the other hand, organic mitral regurgitation may, *pro tempore*, be murmurless. Now, the first difficulty will occur where the orifice is so much dilated that its valve, though perfectly sound, has ceased to be of sufficient size for purposes of complete closure. There are no possible means of distinguishing such regurgitation from that induced by actual disease of the valve; happily, the phenomenon is very rare; and, as the other characters of dilatation must be present in a high degree, the treatment will not err. The second difficulty—that of murmurless organic regurgitation—will arise from feebleness of the backward current, itself depending on temporary general collapse, or weakness limited to the heart; in a short while, the heart's vigor having improved, mitral murmur becomes audible.—Dilatation differs from *chronic pericardial effusion* by the square outline of its dulness; by the distance from the clavicle to the spot of the apex-beat being natural or increased; by the heart's sounds being superficial, and of about the same strength at the præcordial region as at the top of the sternum [1184]; by the dulness not extending above the third rib; and by the total absence of friction-sound, no matter in what position the patient be placed.

1342. *Prognosis.*—Under all circumstances a most serious disease, the danger of dilatation increases directly as the excess of the capacity of the cavities over the thickness of their walls; directly, too, as the softness and flabbiness of the heart's tissue; directly, too, as the general deficiency of tone in the system and impoverishment of the blood. Once dropsy has supervened, life can with difficulty be prolonged by art beyond twelve or eighteen months.

1343. *Treatment.*—Dilatation of indubitable existence is not removable by treatment: those doubtful cases of the disease, which Dr. Hope professed to cure, bear no claim to scientific recognition. But if it be not, in the highest sense, a curable affection—though art cannot remove dilatation, art can render dilatation bearable, and even unfelt. In conducting the treatment, the essential clinical element of the disease, weakness (whether dilatation be primary or secondary), must be constantly held in view, all debilitating measures systematically avoided, and the effort made to improve the heart's tone without exciting its irritability.

1344. Unprovided, as we are, with any medicine exercising a specific action on the tone of the heart's fibre, general tonics must be resorted to. Bitters, mineral acids, and preparations of iron, in doses and combinations modified according to circumstances, supply the groundwork of medicinal treatment: it is of course to be understood that no positive contra-indication to the use of these agents exists beyond the heart. Opium or belladonna, internally and externally, tranquillise excited action better and more safely than other sedatives.* Aconite may be given in very small doses, on urgent occasions, but by no means employed habitually. The exhibition of digitalis requires the utmost caution: slackening the circulation, as it does, it promotes either coagulation within the heart, or, in a less degree of its action, accumulation of blood in the cavities, whereby they may be still further passively dilated. If the power of the ventricles be seriously impaired, digitalis cannot be given without excessive risk, and had much better be altogether avoided. Should the heart be nervously excited, the various anti-spasmodics are indicated. The inhalation of chloroform or æther cannot, under any circumstances, or for any object, be permitted.

The condition of the chylopoietic viscera must be carefully watched,—due action from the bowels ensured daily, without weakening purgation (aloetic medicines are the best), and the action of the liver, if this be sluggish, promoted by taraxacum and an occasional mercurial aperient.

The patient should lead a tranquil life, avoiding all excitement, but mixing in cheerful society; his exercise should be moderate, always considerably within fatigue; very gentle use of light dumb-bells, by promoting the circulation through the upper extremities, seems to act beneficially; but caution is requisite in permitting this. A good nourishing animal diet, of plain, easily digestible character, is that fittest for these patients. Much drink of any kind is to be avoided; bitter beer in moderation is allowable at dinner, or, if the patient have been accustomed to take these, a glass, or at most two, of port or sherry;

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* The influences of belladonna and opium are in some points of view so unlike, so opposed, indeed, that possibly the states of heart and circulation relieved by each, are not so really similar as they appear. See a paper by Dr. T. Anderson, on the Effects of Belladonna in Poisoning by Opium.

dandelion coffee is a good material for breakfast. A dry, bracing air, as a rule, agrees best. Particular circumstances, it is true, may require the patient to live in one of the very opposite characters; but, though a relaxing atmosphere may relieve an accompanying dry bronchitis, it unquestionably tends to depress still further the heart's energy. Flannel next the skin is indispensable; the shower-bath may be cautiously tried, and continued or not according to its visible effects,—effects which, it has appeared to me, are, in all diseases, much under the influence, first, of idiosyncracy, and, secondly, of the patient's previous habits in respect of ablutions. Tepid salt-water bathing, or even, when the affection is not far advanced, quiet immersion in the open sea, is useful; swimming exercise, it is scarcely necessary to say, should be absolutely avoided.

1345. Paroxysms of dyspnœa may be relieved by hydrocyanic acid, cannabis indica, æther, and liquor opii sedativus, and the æthereal tincture of lobelia inflata; by dry-cupping, or the application of three or four leeches to the præcordial region, especially if there be palpitation; or by the use of Junod's exhausting apparatus. If disturbed action be traceable to hysteria, the usual remedies for that state should be given; if to flatulence, carminatives sometimes tranquillise the organ almost instantly. Pulmonary congestion and sub-inflammation require dry-cupping, mustard poultices, flying blisters; and, as they are rarely active, squill and ammonia or senega internally. Should actual pulmonary inflammation occur, antimony must be employed with great caution: it is easier to depress the vital powers than to raise them. Systemic dropsies are removable by the methods elsewhere described [1328]; they should not be too rapidly removed, supposing surgical influence becomes requisite, as the sudden loss of support on the part of the vessels may induce prostration of strength, from which the patient cannot be recovered.

CHANGES OF CONSISTENCE.

SOFTENING.

1346. In addition to cases in which softening of the heart results from acute inflammation, hæmorrhagic infiltration, or fatty disorganization of its texture, that state is met with in certain acute febrile affections—for instance, typhoid and typhus fevers; and in some chronic blood-diseases, as scurvy and purpura. In all these instances, softening constitutes an accident of the primary affection; but there are other cases in which the heart notably loses its consistence, without there being any prominent blood-disease, or any obvious affection of the organ itself, except the deficiency of firmness. To the latter cases alone it is proposed at present to refer: they are, perhaps, more uniformly connected with fatty alteration, than can at the present day be demonstrated.

1347. The impulse of a heart of this kind may be invisible, or visible with some and invisible with other beats, and occasionally somewhat undulatory. If any single beat be of considerable force, the left ventricle is very certainly somewhat hypertrophous as well as soft. Percussion discloses nothing, unless, as is often the fact, the flaccid organ be dilated also. The first sound is short, flapping, weak, toneless, almost to extinction; the second weak and thin: there is no murmur.

The pulse, irregular in force, may be excessively so in rhythm,—no connection of time being traceable between it and the systoles of the heart.* Excessively feeble, fluttering, and small, it is not of necessity very frequent: there are instances, indeed, in which it has appeared unnaturally slow; this may sometimes, but certainly not in all instances, have been deception, from the weak systoles not always impressing the distant vessels.

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* In typhoid fever these characters of pulse, suddenly supervening, have been shown by M. Louis to be significant of *acute* softening of the heart. The observations of Dr. Stokes on the state of the heart in continued fever are full of interest.

1347*. There are few symptoms that can be distinguished from those of dilatation. General languor, weariness, incapacity for exertion of any kind, failure of strength, sallowness and lividity of the face, feeble palpitation, sighing respiration, protracted fits of dyspnœa, and painful sensations about the heart, occasionally actual angina, are common to the two affections. Dropsical effusions probably do not occur unless there be dilatation, in addition to alteration of the blood: but the fact is, softening of any duration is rarely unaccompanied with some increase, general or local, of capacity of the cavities. Softening is always a serious condition: it aggravates the ill effects of any other heart-affection it may co-exist with, and has of itself proved suddenly fatal, with or without symptoms of angina.

1348. The feebleness of impulse, without increased area of dulness, and the feeble irregular pulse, without the signs of valvular, especially of mitral, disease, are the points on which the diagnosis of softening turns; but these characters will not distinguish simple from fatty softening,—and scarcely from dilatation.

1349. *Treatment.*—In the management of the disease, an attempt must be made to improve the nutrition of the heart by raising the powers of the system generally, and improving the blood. Animal food, port wine in moderation, pure bracing air, regular but very gentle exercise, and attention to the state of the skin, are the hygienic means indicated. To tonics, quinine, iron, and mineral acids, we must mainly trust among medicinal agents. Astringents, provided their ill influence on the bowels can be obivated, might be advisable—among the number, gallic and tannic acids.

Quietude, mental and bodily, is indispensable; too much exercise is infinitely worse than none at all; indeed, the patient's instinct, as well as his feebleness, leads him to disobey the instructions, sometimes given in such cases, for forced pedestrian exercise.* Fits of passion should be studiously avoided.

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* It appears to me that error is very frequently committed in the attempt

Direct sedatives of the heart cannot be given without extreme danger; and inhalation of æther or chloroform is under no circumstances permissible.

INDURATION.

1350. General induration of the heart, excessively rare under any circumstances, scarcely occurs except from contractile hardening of solidified lymph and calcification. It may be supposed to weaken the muscular, and intensify and give undue clearness to the valvular, portion of the first sound. Should any knocking sound exist at the apex, it would probably be peculiarly clear. But these signs require revision at the bed-side: I give them hypothetically, having never met with a case of the kind.

1351. Induration of the papillary muscles of the left ventricle is not very uncommon: if combined with contraction of their substance, it must cause mitral regurgitation. The induration-matter infiltrating them has occasionally been calcified and even pseudo-ossified.

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to force patients, labouring under various diseases, to *walk, walk, walk,* in spite, often, of their own convictions that each additional walk has helped them on towards that bourne, whence no walker returns. I do not refer, merely, to the bygone well-known practice in a central provincial town,—but to the too indiscriminate recommendation of toilsome foot exercise by many of the profession generally. I have known a man labouring under chronic nephritis, with alkaline urine and the general cachectic condition of that disease, directed to perform such pedestrian feats, by way of recovering his health, as he himself well knew would, on the very first day, have brought on such an acute attack, as probably must have confined him to bed or a sofa for a month. I entertain not a doubt that the lives of sufferers from cardiac disease are often put in jeopardy by the effort to exercise beyond their powers. Because, in the *physiological* order of things, it is good to walk, the inference is at once drawn that it is universally good in the *pathological* order of things. But, in point of fact, each disease has its own appropriate kind and quantity of exercise,—and that kind and that quantity are, as all truths in therapeutics, to be learned in each instance by *experience* alone.

ADVENTITIOUS PRODUCTS.

CALCIFICATION.

1352. Calcification may occur in any one of the tissues of the heart, in the actual substance either of induration-matter, fibrinous coagula, atheroma, or tubercle. In the valves it produces obstruction and regurgitation; in the cavities its effects coincide with those of polypoid concretions; in the pericardium, and still more in the heart's substance, calcification obstructs the heart's movements; in the coronary arteries, if carried to any extent, it must interfere with the nutrition of the organ, and may induce fatal angina.

1353. Calcification of induration-matter in the pericardium may conceivably give an osteal quality to the percussion-note over the heart, and impress something of a similar character on the systolic sound: here are conditions which, with a previous history of pericarditis, might give an inkling of the existence of the state in question. I have seen one example of lamellar pseudo-ossification of the pericardium. There are no known means of distinguishing calcification of the endocardium from other morbid changes, interfering organically with the play of the valves.

FATTY DISEASES.

I.—FATTY INFILTRATION.

1354. Fat accumulates in three positions in connection with the heart—(*a*) under the pericardium, (*b*) under the endocardium, and (*c*) amid the muscular fibres.

1355. (*a* and *c*) Sub-pericardial fat, chiefly gathering about the right side of the organ, and the ventricles rather than the auricles, when abundant, encroaches on, and to a greater or less depth renders the muscular texture beneath soft and atrophous. But this influence on the nutrition of the fibres is produced by simple pressure, not by intrinsic deterioration of the fibre itself.

1356. Laennec was of opinion that this species of fatty affection "must exist in a very great degree before it gives rise to any serious complaint:" and he was in all likelihood right. True, cases have been recorded in some number, where the gravest effects have been assigned to such fatty disease; but very probably it must either in these instances have led to total destruction of fibre in limited places, or intra-sarcolemmous fatty change must have coexisted. There are no recorded proofs, as far as I know, that rupture of a fatty heart has ever taken place, unless where the latter condition existed; though the accident very conceivably may have occurred independently of this.

1357. Still fatty accumulation under the pericardium produces minor annoyances, and some physical signs. Sensation of oppression, or even pain, about the præcordial region; syncopal feelings on exertion; inability to walk quickly on level ground and to get up hill, except with great and painful effort; inclination to coldness in the extremities; feeble (but, as far as I positively observed, regular) pulse, of about medium frequency; sluggish action of the liver and bowels; occasional giddiness; and feeble cardiac impulse, with a too extensive dulness under percussion, the sounds, especially the first, being weak and toneless:—these were the symptoms and signs in the only person, a male, aged sixty-four, I happen to have watched professionally during life and opened after death, whose heart was at once loaded with sub-pericardial fat, and positively free from serious softening or notable amount of intra-sarcolemmous oil. There was in this instance a considerable quantity of fat in the lower part of the mediastinum, which may have contributed to weaken the heart's shock against the side. Dr. Hope speaks of the pulse being irregular in such cases; but in the only instance where he made a *post-mortem* examination, the tissue of the organ was softened as well as loaded with fat.

(*b*) Minute pellets of fat are sometimes seen under the endocardium: in the present state of knowledge they are devoid of clinical interest. I have also frequently noticed microscopically, fat, granular, and oily, between the laminæ of the aortic valves; and also in the substance of fibrous vegetations on the free surface of various valves: but the clinical import of these conditions has yet to be worked out.

II.—FATTY ATROPHY.

1358. A fatty pseudo-metamorphosis of the muscular fibre of the heart, or rather deposition of fat, sequential to the atrophous destruction of, and in place of, that fibre, is the most important affection belonging to the present class.

1359. Laennec acutely saw the distinction between this form of destruction and mere fatty infiltration, with muscular wasting produced by pressure, and to his description of the naked-eye appearances modern observation adds but little. Parts of the organ thus affected lose consistence more or less, tearing in extreme cases with the greatest ease; vary in tint from a delicate faded-leaf hue to a pale dirty brown, mottled with darker spots; rarely feel greasy, though readily yielding oil under pressure; the organ, in most instances natural in size, may be hypertrophous, or, as is much more common, dilated; locally infiltrated with blood, aneurismal or actually ruptured. The coronary arteries may be atheromatous, calcified and obstructed, or healthy; nor does there exist any necessary relationship between morbid conditions of these vessels and the muscular waste. The aortic or mitral valves, or both, are sometimes diseased; but there does not seem, from what has fallen under my notice, to be any necessary, or even close connection between the two states; neither does calcification of the aorta appear to be more frequent than in persons of the same age, cut off without fatty heart.

Both ventricles are most commonly affected; the left alone more frequently than the right alone. The layers of substance immediately beneath the endocardium and the pericardium suffer earlier than the intervening strata; eventually the entire thickness of the walls, papillary muscles, and columnæ carneæ, become involved. The auricles seem very little prone to this morbid change: Dr. Ormerod questions its ever occurring there; but I have most certainly observed it in the right auricle.

1360. The microscopical characters of the disease are closely the same in the heart as in voluntary muscles. But its very earliest evidences are perhaps less readily caught; because, as Dr. Ormerod observes—an observation in which I concur—the transverse striæ are in the normal state less distinctly marked in the cardiac than in the voluntary fibre: this, at least, I hold to be true of the adult heart. The outline of the fibre is preserved, while the striated appearance wholly loses its clearness of definition, a granular condition taking its place; little by little the granular appearance, at first even and transverse, becomes perfectly irregular; meanwhile oil-globules accumulate within the sarcolemma more and more, till they completely obscure any remaining muscular element. Oil is also found outside the sarcolemma, probably in part produced there, and in part reaching that situation through rupture of the sarcolemma. Æther, dissolving the oil, will disclose elements of fibre, where they had previously been imperceptible. The diseased fibres are not notably, if at all, less in diameter than healthy ones.

1361. The disease has been noticed in the subjects of old pericarditis and endocarditis,—whether as a coincidence, or as an effect, remains to be proved. So, again, it has been found co-existent with phthisis, and various pulmonary inflammations, with scurvy, purpura, renal diseases, gout, chronic rheumatism, paraplegia, hepatic disease with gall-stones, hæmorrhage in various situations, and continued fever,—in a word, there seems to be no diathesis which excludes it, nor is it protective against any other form of disease. On the other hand, fatty disorganization may be the sole affection in the body.

1362. It would be foreign to the purposes of a purely practical work to discuss the mechanism of fatty degeneration,—as exposed variously by Paget, Ormerod, Quain, Lehmann, Valentin, and Handfield Jones. But it may be as well to state my belief that the recognised laws of vital processes, as well as attainable positive evidence, are repugnant to the admission of the direct metamorphosis of protinaceous into fatty matters in the midst of a tissue living and active,—as of so much dead muscle into adipocere. I agree with those who infer that, first, atrophy of the sarcous elements occurs, and that, secondly, oil is molecularly laid down in the place of the absorbed elements. The fat seems the mere index, in no wise the essence, of the disease.

Hence the true problem becomes the determination of the causes of the primary failure of nutrition of the sarcous structure: and of this we are utterly ignorant. The whole history of this and allied affections deposes against its merely local mechanism;* while, on the other hand, we are unacquainted with any form of blood-disease or nervous dyscrasia even seemingly essential to its existence.† And admitting the reality of some, as yet undiscovered, preliminary morbid state of the blood, it would remain to be ascertained why that particular form of pravity of the fluid should lead to the impairment of the nutrition of the heart rather than of other textures. The disease is fundamentally different pathologically from, however closely allied clinically to, mere fatty infiltration.

1363. Though occurring in youth, fatty disorganisation is essentially a disease of middle and advanced life; most common in males; occurs in all ranks of society, though most frequently perhaps in the middle and upper; and in rare instances may be seen in individuals cut off by acute disease, while systematically living after the most approved hygienic models. The influences, if any, of manner of life, and quantity * and quality of food are unestablished.

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* The fatty metamorphosis of muscles adjoining non-reduced dislocations and ankyloses, and that occurring in paralysed limbs, and in bed-ridden people, is explicable by the waste consequent on inaction: but it is difficult to admit even the minor influence of any such cause in the case of the heart.

† Dr. Smith of Dublin, in some of these cases found oil in the blood; but can we, with Dr. Stokes, accept the fact as an "apparent proof" that fatty heart may be produced by the assimilation of ready formed oil in the blood? I am disposed to think not: the two conditions were probably mere coincidences. Piarhæmia may exist without fatty heart, and fatty heart without that diseased state of the blood.

1364. The clinical aspects of the disease vary greatly with the superficial extent and the degree of the atrophous change. That fatty metamorphosis may be found, and to no insignificant amount, where neither subjective nor objective cardiac symptoms had awakened attention during life, is indubitable. And it is equally certain that hearts have been carefully examined during life, and pronounced free from disease, which almost immediately afterwards (death arising from other causes) have been found very sensibly fatty. The disease may then to the observer of the present day, at least in its minor and moderate degrees, be *latent.*

1365. *Signs and Symptoms.*—But the rule is that, if the affection be at all marked, its clinical evidences are more or less precise and positive.

1366. The physical signs are those of a soft heart: weak impulse, indistinctness of the apex-beat, percussion-dulness, both superficial and deep, unchanged in area, unless there be alteration of bulk from some independent cause; a feeble, toneless, short first sound; a long first silence, and a feeble, but relatively distinct and accentuated, second sound. If the fatty disorganisation be, as it often is, in great excess in the left ventricle, the first sound may be of notably fuller tone at the right than the left apex, and the second sound louder and more strongly accentuated at the second left, than at the second right, cartilage: the relative state of preservation of the muscular structure of the two ventricles explains these facts. The indistinctness of the apex-beat is probably owing to the want of power of the fibres, naturally giving the spiral movement to the heart in systole: the whole organ is simply, and as it were lazily, projected forwards. Possibly a dynamic mitral regurgitant murmur may sometimes occur; but I do not know this from observation; and, in the present state of knowledge, if murmur exist, its explanation must be looked for in valvular disease or alteration of the blood. I have never met with absolute deficiency of either sound over the entire cardiac region.

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* Aristotle ascribed conversion of muscle into fat to excess of nourishment.

1367. The symptoms in highly marked cases are somewhat as follows. The patient, though not necessarily out of health generally, unable to undertake any sustained labour, exhausted on the first attempt almost, forced often, when making an effort to walk, to stop at every few paces from cardiac uneasiness, dyspnœa or both, is irritable in temper, dejected and peevish,—emotional states coming either directly of the disease and its perverted state of circulation, or, indirectly, of the incapacity for occupation it entails. His tissues generally are soft and flabby; his muscular power almost null; the skin of the face pale and sallow, or more or less livid; the feet and ankles disposed to œdema; the appetite feeble, the digestive forces languid, the bowels confined, the liver larger than natural, and readily increased in size during paroxysms of dyspnœa.

Easily put out of breath, as a rule, and subject to fits of dyspnœa of very irregular rhythm and sometimes orthopnœal, yet not constantly asthmatical, the patient may, on the contrary, be free, even under great cardiac distress, from subjective or objective difficulty of breathing. In rare instances the breathing exhibits very peculiar characters, thus described by Dr. Stokes: a series of inspirations takes place increasing to a maximum, and then declining in force and length, until a state of such complete apnœa is established, that the patient may be supposed dead, when a low inspiration marks the commencement of a new ascending and then descending series of inspirations; the patient does not suffer during the suspension of breathing, and there is no physical sign except respiration weak to suppression, followed by its most highly exaggerated form.* A tendency to sigh, without emotional influence or mere habit to explain it, may in some cases be connected with the commencement of fatty metamorphosis. I have known cough a troublesome symptom, unattended with signs of bronchitis or any other pulmonary affection.

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* Dr. Cheyne observed this phenomenon in 1816: each revolution in the

The pulse may be perfectly regular in force and rhythm, and of medium frequency, as the habitual state, even where strong evidence exists of advanced fatty change,—and in one case of this kind, even when the pulse became paroxysmally frequent, with anginal pain, it maintained its regularity. But this is the exception. Commonly the pulse is irregular in force and rhythm, either constantly or from time to time under the influence of excitement, flatulence, indigestion, or effort. On such occasions it may become exceedingly frequent: I have known it uncountable—in the main from frequency; in part, however, from irregularity. Infrequency of pulse, occasionally met with, is in some cases referrible to the weakness of occasional systoles; but the systoles are themselves sometimes much less frequent than natural, and may fall, as originally noticed by Dr. Adams, to thirty, or even less, in the minute [1145]. The pulse may, when frequent and irregular, have also a tingling character, according to Dr. Stokes: but in the only instance, he reports, there was marked mitral coarctation. What the conditions are, specially productive of these varieties of pulse, remains to be determined; the greatest irregularity may exist without mitral disease, but not, I am inclined to think, without dilatation. Occasional fits of palpitation, attended with choking sensations, cardiac uneasiness, pain or actual angina, occur; and the patient, readily becoming faint on the least exertion, falls into a state of actual syncope from time to time.

Sexual inclination and power both fail: in one of the best marked cases of the disease, clinically speaking, I ever saw in a person under the age of forty-five, it was this failure, and not the cardiac symptoms, that led to my being consulted.

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state of breathing in his patient occupied about a minute; the cessation of breathing, a quarter of a minute; the number of respirations in a minute equalling about thirty.

The encephalon suffers too. Vertigo, failure of sight, dull aching sensations in the head, feebleness of intellect, and especially of memory, and somnolence become common, with the advance of the disease, either as almost habitual states, or as paroxysmal occurrences. And such attacks may proceed farther; the patient, becoming suddenly comatose, may exhibit many of the appearances of an apoplectic seizure,—sudden fall to the ground, complete insensibility with stertorous breathing, while the pulse becomes, even if previously an infrequent one, still more infrequent. The attacks are generally brief, easily recovered from, and very rarely followed by paralysis. The intimate nature of these seizures remains to be unravelled; that the brain is organically sound, even, is far from being demonstrated,—but in their immediate mechanism they appear to me, with Dr. Stokes, rather to depend on deficient arterial supply and venous stasis, than on excess of blood transmitted to the brain. Dr. Stokes refers to a case in which the patient, warned by certain premonitory symptoms, was enabled to save himself from attacks by "hanging his head, so that it rested on the floor." There seems, indeed, a close alliance between these apoplectiform attacks and syncope; and a patient who has long been the subject of syncopal attacks, may lose these and have the apoplectiform variety instead. I have twice observed in cases of fatty heart, as far as clinical evidence is concerned, formication, slight numbness and tendency to feeling of cold in the lower extremities, the patients being free from other ordinary symptoms of disease of the cord, and certainly free from intra-pelvic affections, enlarged prostate, stricture of the urethra, renal disease and spermatorrhœa. Were not these symptoms of implication of the medulla analogous in mechanism to the quasi-apoplectic seizures?

Little or nothing is known positively concerning the state of the blood. I have failed to find the venous hum of anæmia, in cases where the tissues were markedly pallid,—but the patients were aged. The piarhæmia, noticed by Dr. Smith, was clearly exceptional and accidental. It is more than doubtful that the urine possesses any distinctive, or indeed habitually morbid, character: I can answer in the negative for fat, albumen,* sugar, biliphæin, excess of urea, casts of the tubules, and spermatozoa; oxalates, uric acid, and amorphous and triple phosphate, occasionally present, are of no significance.

1368. *Prognosis.*—Patients labouring under this disease, are often possessed with the idea, which occasionally proves a prophetic one, that they shall expire suddenly. Death has, in truth, occurred instantaneously, either from rupture into the pericardium, or by coma, or by syncope; in other instances, the fatal event is slowly brought about by asthenia. On the other hand, fatty disorganisation of the heart is by no means necessarily fatal: I have known extensive destruction of the kind exist, where death had occurred from unconnected chronic disease of other organs; and the proofs that a moderate amount of the change may be comparatively innocuous are of daily occurrence. Nevertheless, could we positively diagnosticate the disease, no matter how slight its extent, its existence should never be lost sight of, either in regard of prognosis or therapeutics.

1369. *Diagnosis.*—If weak cardiac action, feeble toneless sounds, absence of murmur, and percussion-dulness of normal area, coexist with frequent and irregular or infrequent and regular pulse, respiration of ascending and descending rhythm, and the peculiar cerebral attacks described, there can probably be little doubt of the existence of this form of atrophy of the heart. But the value of this proposition is less than it seems; for, even in cases anatomically well marked, the pulse may be perfectly natural; apoplectiform attacks are very rare; and the perverted rhythm of respiration seems only to occur within the last few weeks or days of existence, when the question of diagnosis has lost its chief practical utility. The possibility of infrequency of pulse being a normal state, too, must not be forgotten [1144]. Besides it remains to be proved that certain forms of dilatation are not capable of producing these cerebral and respiratory disturbances. The physical signs enumerated, coupled with the deficiency of the dropsies and other systemic effects of dilatation, seem to furnish the most serviceable guides to direct diagnosis.

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* Slight albuminuria occurs where dilatation coexists with fatty change; but I have not known the latter, unaided, produce it.

Mr. Canton shows the arcus senilis to be the result of fatty atrophy of the cornea. But the arcus senilis, like fatty heart in the generality of cases, is an attribute of age: its discovery will do no more in confirmation of an otherwise doubtful diagnosis of fatty heart, than would ascertaining that the patient has passed his fiftieth year. If indeed the corneal change were found in a youthful person, it might possibly have some weight: that fatty heart may exist without change in the cornea is certain,—and the converse holds equally good.

The tendency to infrequent pulse perverts the pulse-respiration ratio,—but observations are wanting on this point.

1370. Of the impossibility of distinguishing simple from fatty softening I have already spoken [1348]; but, fortunately for purposes of diagnosis, the simple change is of extreme rarity; nor do I know of any means whereby fatty pseudo-metamorphosis may with surety be distinguished from mere infiltration. The researches of M. Bizot into the relationship of subcutaneous fat to fat-accumulations about the heart furnish no clinically available help. The natural areæ of the cardiac dulnesses, and the absence of systemic congestion and dropsy will distinguish fatty disorganisation from dilatation; if there be marked jugular distension, or *à fortiori*, jugular pulsation, the case is not one of fatty disease. From all forms of valvular disease the present affection differs in the absence of organic intra-cardiac murmur: still a dynamic mitral regurgitant murmur possibly may exist; basic systolic murmur of blood origin is rare; whatever be the morbid state of the fluid in these patients, it seems unfavourable to murmur. From chronic pericardial effusion fatty disease will differ by the normal area and shape of cardiac dulness, by the absence of præcordial arching, and by the fact that the heart-sounds are at least as well marked at the cardiac region as at the top of the sternum; the history of the case will prevent the possibility of error.

1371. The apoplectiform seizures are distinguished from epileptic attacks by the absence of convulsions, of foaming at the mouth, and of cervico-facial turgescence. But at the time of seizure it may be much more difficult to distinguish them from uræmic apoplexy. The deficiency of marked changes in the urine and of anasarca will be the fundamental guides to distinction. In the pallor of face and absence of subsequent paralysis the two varieties agree.

1372. *Treatment.*—The treatment of this affection is essentially that recommended for simple softening of the heart. It seems natural to enjoin abstinence from fats of all kinds, and of materials amylaceous and saccharine, readily converted into fat; yet there may be little philosophy here after all,—if the local fatty state be, as seems strongly probable, a mere evolution of a form of atrophy. But protinaceous food is unquestionably proper, in full, but divided, quantity; the moderate use of wine, or brandy, in no wise evil.

The sufferer from fatty heart should be very cautiously submitted to lowering regimen or depressing medicinal treatment for any intercurrent attack that might, in a previously healthy person, call emphatically for such measures. Chloroform to the præcordial surface even is inadmissible: its inhalation hazardous to the last degree, though it is difficult to avoid the conviction that people with fatty hearts have occasionally inhaled with impunity.

The gentle but steady use of electro-galvanism seems to me seriously worthy of trial; physiological results are, however, very contradictory on the point.

Moderate exercise on level ground, if not productive of distress, should be daily taken. Pure bracing air is theoretically indicated. But I am unacquainted with any facts proving that forced exercise in mountainous countries can cure, or even stay the onward progress of, the disease we have been considering; the cardiac affections I have known removed by Alpine tours have been simply functional derangements of the heart dependent on gastro-hepatic dyspepsia.

The apoplectiform seizures, intercurrent to the disease, should clearly be met by stimulants internally and counter-irritation to the lower extremities: they ought in fact to be treated much as syncopal attacks.

TUBERCLE.

1373. (*a*) Tubercle very rarely forms in the heart; and only in cases of general tuberculisation, or as an extension from similar substance deposited under the pericardium. Its signs and symptoms, if it have any peculiar to itself, are unknown.

1374. (*b*) More commonly, tubercle forms beneath the serous layer of the pericardium, either cardiac or parietal. Occurring in the state of semi-transparent gray granulation, it may remain latent, or excite inflammation of that membrane. The ensuing pericarditis produces the same physical signs and local disturbance as if it were of idiopathic origin. But, probably on account of the tendency to secondary formation of tubercle amid the simple inflammatory exudation-matter,* the disease, unless fatal at once, tends just as peritonitis of the same origin, to lapse into a chronic state, attended with more or less constant local symptoms. This was the view of Laennec; and it is supported by the course of a case that fell under my own notice.

1375. In ordinary phthisis, as appears from the records of M. Louis, tuberculous pericarditis is very rare: it is greatly more uncommon than pleuritis or peritonitis, and notably so even than meningitis, of that form.

1376. The diagnosis of tubercle in the pericardium can only be made through the pericarditis it entails; true, it is infinitely probable, that miliary tubercules would themselves produce friction-sound, but in the present state of knowledge, the discovery would rather lead to a mistaken than correct diagnosis. And the tuberculous character of the pericarditis would not be announced by any peculiarity in its signs; still, an attempt at divining its character might be made in the following way. If there were neither rheumatism, nor Bright's disease, nor adjoining pleuro-pneumonia present, and the patient had received no injury to the chest, and was distinctly of strumous constitution, the probabilities would be much in favour of the tuberculous origin of the inflammation. And this, too, although there were no distinct pulmonary signs of advancing phthisis; for, herein resembling tuberculous peritonitis, this form of disease has mainly been observed in cases where the lungs were but slightly affected.

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* Carswell's Drawings, U. C. Museum, A. 533, Fig. 2.

1377. The determination of the nature of such pericarditis is not a mere matter of diagnostic curiosity. The most appropriate treatment, as Dr. Burrows has successfully argued (Med. Chir. Trans., vol. xxx.), can scarcely be that adapted to rheumatic pericarditis. Blood-letting should be had recourse to in extreme moderation; mercurials must be given with all needful care to avoid ptyalism. Blisters in the neighbourhood of the præcordial region, dry-cupping, moderate purgation, diuretics with iodide of potassium and alkalies, are the agents on which, in connection with antiphlogistic regimen, reliance must be placed. Animal food may be allowed earlier in this than in other varieties of the disease.

CANCER OF THE HEART AND MEMBRANES.

1378. Cancer occurs in the heart primarily or, much more commonly, secondarily; in the nodular and infiltrated forms; and of encephaloid chiefly, scirrhous occasionally, species. Eleven-twelfths of the entire organ have been found implicated; at the other extreme we have examples of a single pea-like mass.

Nodules may form under the endocardium and pericardium, without actually implicating the heart's fibres.* Cancerous destruction of the aortic valves, and also extensive cancerous accumulation in the pericardial sac have been observed.

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* Clin. Lect., Subcutaneous Cancer, *Med. Times*, Aug., 1852.

Carditis has not been noticed in any recorded case; recent endocarditis, old pericarditis, and dilated hypertrophy are described as coexistences. Cancer of the heart does not spread to other organs, but cancer of the lung and mediastinum has encroached upon the heart.

1379. Cancer of the heart, greatly more common in the male than in the female sex, has been met with at all ages.

1380. In a case recorded by M. Andral, where the morbid growth had almost completely taken the place of the muscular structure on the right side of the heart, slight dyspnœa was followed after five months by a sudden and temporary attack of severe præcordial pain, increased dyspnœa, vomiting and loss of consciousness; the succeeding year the habitual dyspnœa increased, and seven or eight paroxysmal attacks, such as just described, occurred; six weeks before death emaciation and straw-coloured tint of skin were observed. In the intervals of ease nothing could be detected by auscultation. Anasarca, commencing with the lower extremities, reached the upper limbs and the face; and the patient died suddenly without struggle.

1381. Cancer under the pericardium will give no friction-sound, unless the corresponding serous surfaces be rough, or at least dry. [472].

ENTOZOA OF THE HEART.

1382. Entozoa infest the heart sometimes,—both the acephalocyst and the cysticercus. An acephalocyst the size of a pigeon's egg is seen in the septum of the ventricles in the heart of a woman (U. C. Museum, No. 2293), who died suddenly while engaged in her household affairs. Among Dr. Carswell's drawings (U. C. Museum, A. 9), is the figure of a heart containing in the posterior part of the left ventricle a full-sized acephalocyst, which protrudes on the surface. The patient, a female, was cut off by phthisis, and the entozoon accidentally found on the dissection of the body; and as she had died in hospital, and the heart had not attracted attention, there were, probably, no symptoms.

DISEASES OF THE ORIFICES OF THE HEART.

1383. *General Effects.*—Diseases of the orifices of the heart interfere in either, or both, of two ways with the circulation through the organ. They are constrictive in anatomical character, and hence produce *obstruction* of the onward current; or they entail disproportion between the size of the orifice to be closed and the valve to close it, and hence lead to *regurgitation* of a backward current.

1384. Each orifice is in theory capable of each kind of disease,—obstructive and regurgitant; but the frequency with which the various orifices actually suffer differs very widely. This question, however, requires to be considered in two points of view, the anatomical and the clinical: the excess of organic change in the actual texture of the valves and orifices of the left side, is very great; the excess of disturbance, clinically demonstrable, in the functional conditions of the orifices of the left side, much less. This comes of the frequency with which the tricuspid valve is incompetent, through mere widening, without textural change, of the orifice, while its own structure remains sound: the excess of textural disease of the valves on the left side is counterbalanced by the excess of misfitting of the closing valve on the right.

1385. Obstruction of orifices, as far as is known positively, is always of organic character, and is caused by morbid change in the valves, in the wall of the orifice itself, or by extraneous pressure. Regurgitation through orifices is, certainly, in the vast majority of cases, likewise of organic character, and is caused by morbid change in the valve, in the wall of the orifice, or in some part of the apparatus connected with the closure of the valves. But it is next to certain that regurgitation may be produced dynamically in the mitral orifice at least, through functional imperfection of the closing apparatus, though this be perfectly free from organic change: regurgitation of this kind is, however, of temporary duration, and does not produce results of importance. It is possible, too, that obstructive disturbance may be produced at the mitral orifice by perverted action of the papillary muscles; and either regurgitation or obstruction at the mouth of the aorta by perverted innervation of the sygmoid valves: but all this is completely hypothetical at the present day; and hence, practically, the serious diseases of orifices may be regarded as organic and statical.

1386. Diseased valves, diseased orifices, and diseased states of the apparatus effecting the closure of the valves, are, then, the causes of perverted circulation through the outlets and inlets of the heart.

(*a*). *Diseased valves* are the causes of *obstruction:* first, through local endocarditis and its products interstitial and superficial, alterations of form, and adhesions of the divisions of the valves *inter se;* secondly, through deposits of fibrine from the blood on their surfaces; thirdly, through atheroma, calcification, and their sequences; and, fourthly, in some instances, through hypertrophy. Diseased valves, again, are the source of *regurgitation:* first, through endocarditis and its effects (especially when these tend to produce puckering and diminished superficial size, or actual destruction of the divisions of the valves, or adhesion of these either to each other, or to the adjacent wall of the heart or great vessel to which they belong, or to produce thickening and shortening of the chordæ tendineæ, or rupture of these); secondly, through fibrine deposited among, and interfering with, the action of the chordæ tendineæ; thirdly, through atheroma and its sequences; fourthly, through hypertrophy of the valves interfering with the freedom and completeness of their fall at the proper moment; fifthly, through atrophy of the valves, affecting their depth or superficial extent, in rare instances affecting their continuity by extensive perforation; and, sixthly, through atrophous shortening of the chordæ tendineæ.

(*b*) *Diseased orifices*, the actual substances of the valves being sound, lead to *obstructions* through the products of endocarditis, atheroma, and calcification lying upon, or producing changes in, their surfaces or walls. And disease of the orifice is the cause of *regurgitation*, where the valve being sound, the opening is morbidly widened to such an extent as to render the former inadequate to its closure.

(*c*). *Diseased states of the apparatus*, connected with the closure of the valves, are seen in induration and diminished bulk of the papillary muscles, and shortening of the cordæ tendineæ; they produce *regurgitation.* I once met with a direct mitral murmur, where the orifice, instead of being obstructed, was actually too wide; a corda tendinea from the long tongue of the valve bifurcated, each division attaching itself to a separate papillary muscle, and so placing a thread-like obstruction in the way of the current.* It may be made a question, too, whether imperfect action of the papillary muscles, from textural disease of their substance, may not act obstructively also, and throw an obstacle in the way of the passage of the blood from the auricle to the ventricle.

1387. Such are the general characters and immediate effects of the morbid states of the orifices and their connected apparatus. We will now pass to the consideration of the signs and symptoms of these morbid states at each orifice. These signs and symptoms, it must be noticed, are solely of mechanical origin; and hence their discovery discloses the existence simply of obstruction or regurgitation at a certain orifice, but tells nothing directly, and by necessary inference, as to the nature of the morbid process, whether inflammatory, fatty, calcifying, fibrillating, exudative, ulcerative, or cancerous, that has engendered the mechanical difficulty. In the majority of cases valvular disease entails some form of anatomical change in the substance of the heart itself; the proper signs of the former and of the latter are, therefore, met in frequent clinical association; for purposes of precision, it is necessary each class of signs should be enumerated apart,—as is done in the following descriptions. As already shown, too [454], different valvular affections often co-exist, and in certain combinations the one may tend to throw into the shade, or actually modify, the signs of the other or others.

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* Pickett, U. C. H., Males, vol. vi., p. 231.

1388. To save the necessity for repetition with the account of each valvular disease, it may be stated here, once for all, that valvular disease *per se* never alters the area or intensity of the heart's dulnesses, superficial or deep-seated. If either be affected, the presence of some other morbid state becomes matter of certainty.

MITRAL REGURGITATION.

1389. How is the reality of mitral regurgitation during life to be demonstrated after death? Tie the aorta and coronary arteries, cut off the apex of the heart, and let fall against the mitral valve a full-sized column of water; the valve will either support the column or allow it to pass through to the auricle. If the whole of the ventricle above the floor formed by the valve remain filled with the water, scarcely a drop filtering through, there can be no doubt the valve was competent during life.* If, on the other hand, the water escape rapidly, the incompetence of the valve may be held certain. But in the case of slight filtration through the orifice, it would scarcely be justifiable to infer the insufficiency of the valve during life, as we have no means of imitating the vital contractions that accompany the act of closure in the living and moving heart.

1390. *Direct Physical signs.*—The impulse, in highly marked disease of this species, is irregular in force and rhythm. Systolic thrill may sometimes be felt at and about the left apex.

But the essential character of this regurgitation is systolic murmur of maximum force at the left apex, and possessing the other characters already enumerated [445]. The systolic sound, completely or incompletely covered in this position by the murmur, may be perfectly natural at the ensiform cartilage and at the mid-sternal base; the second sound often weakened, in consequence of diminished calibre of the aorta, at the second right, or aortic, cartilage, is accentuated in many, but not in all, instances at the second left, or pulmonary cartilage. Sometimes the second sound is distinct and sharp at the left apex, much sharper than at the pulmonary cartilage. (See case of Anne Gippin [1394].)

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* Such was the fact in Wilkinson, U. C. H., Males, vol. ix., p. 306; Landers, U. C. H., Females, vol. ix., p. 289; Fosbury, U. C. H., Males, vol. x., p. 133; Doyle, U. C. H., Males, vol. x., p. 238.

Systolic murmur limited to the left apex is never of blood origin, as far as I have observed, therefore never chlorotic: the existence of chorea will distinguish the peculiar dynamic murmur of that disease: the murmur of this site and rhythm attending simple dilated hypertrophy will commonly disappear under treatment. The distinction of murmur caused by mitral regurgitation and that produced by friction of blood against irregularities of surface at the base of the ventricle, cannot, I think, be made with positiveness in the present state of knowledge (*vide* p. 247)—but fortunately the latter kind of mechanism is excessively rare. I have already spoken of the influence of feebleness of the heart in preventing the evolution of murmur, of which the physical causes exist: when the heart is texturally feeble, as in fatty softening, its action may be excited by a few turns in a room, or by any other effort, and the murmur will then become audible; when it is feeble from collapse, this must be allowed to pass away, before a diagnosis is ventured on.

1391. *Indirect Physical signs.*—Dilated hypertrophy of the left ventricle is the common sequence of mitral regurgitation; hence the apex-beat and impulse generally are carried outwards, and lowered somewhat. The impulse is increased in force, and in rare instances there is auricular impulse at the second interspace,—either pre-systolic, when it comes of hypertrophy of the auricle, or systolic, when it is communicated from the ventricle. The area of percussion-dulness is increased, especially to the left: but, from sequential hypertrophy of the right ventricle, which I have oftenest observed in early youth, extension of dulness may eventually take place to the right also.

1392. *Pulse.*—The pulse presents itself in either of the two following states:—Regular in force and rhythm; small, with occasional sharpness; rather frequent, and compressible, unless there be much hypertrophy: or irregular in force and rhythm, sometimes to an excessive degree, small, feeble, with occasional sharpness, and tremulous under excitement of the heart. In either of these states, the systoles and radial pulses may fail to correspond in rhythm and in number.

1393. *Effects on the Capillary systems.*—Pure mitral regurgitation produces but little effect on the systemic capillary circulation. The disease may exist for years without inducing either general dropsy or systemic congestion. If dilated hypertrophy supervene, systemic obstruction occurs with a facility proportional to the amount of dilatation and, especially, the impoverishment of the blood. The symptoms then become those of dilated hypertrophy, aggravated, *plus* certain others, to be by and by mentioned, more or less peculiar to the regurgitation. I use the word "aggravated" advisedly; it has not occurred to me to observe cases justifying the fanciful idea that mitral regurgitation may act as a sort of safety-valve to a dilated and hypertrophous left ventricle.

The disturbed conditions of the sensorial functions sometimes observed in mitral disease are likewise the results rather of the dilated hypertrophy with which it is associated, than of itself alone; still, when carried to a great amount, it may secondarily, through its influence on the pulmonary circulation and the right heart, tend to congest the brain passively. No cases have fallen under my notice supporting the notion that colourless softening of the brain is a direct dependence on mitral regurgitation,—a notion very ingeniously advocated by Dr. Law.

The essential effects of mitral disease are pulmonary,—congestive and irritative. The current thrown back through the auricle on the pulmonary veins tends to congest the lungs; and it is not unreasonable to admit that the effort on the part of the right ventricle to overcome the increasing obstruction may induce irritation. Cough, watery expectoration, dyspnœa, and orthopnœa; actual bronchitis, pulmonary œdema, pneumonia, either congestive or irritative, passive congestion and pulmonary apoplexy, are the direct effects of the disease. The expectoration may by the latter condition be stained of dark blood tint, or blackish; or actual escape of pure blood may occur: what maximum quantity of blood may be brought up from this cause at a time, I do not know; I have never seen any large amount.

MITRAL OBSTRUCTION.

1394. *Direct Physical signs.*—I have never observed diastolic thrill at the left apex, though it is conceivable that, if a highly hypertrophous left auricle lie behind the constricted orifice, the current may be rendered sufficiently strong to produce that thrill [365]. The impulse is irregular and unequal in force. The characters of the murmur of mitral obstruction will be found elsewhere [449]; its frequent absence renders the positive diagnosis of this form of disease far from easy. I have already stated that the rhythm of the murmur is rather post-diastolic or præ-systolic, than actually diastolic; I have met with no satisfactory proof of its ever being systolic, as has been affirmed in some quarters,—the evidence given, in such alleged instances of systolic constrictive murmur, to show the absence of regurgitation, being besides, in my mind, insufficient.

Even where a hypertrophous auricle lies behind a constricted mitral orifice, no murmur necessarily ensues, if the constricted surface be smooth, nay even if it be rough,—this is proved by the following excerpt:—

> "Anne Gippin, ætat. 35. . . . p. = 144, regular in rhythm, irregular in force, feeble, small; r. = 48; anasarca; ascites; jugulars swollen, pulsatory; radials faintly visible at wrist; heart's action too extensive,—impulse slapping rather than heaving; faint thrill at apex-region. At mid-sternal

base both sounds dull; first slightly murmur-like, and accompanied by knock; at the left apex systolic roughish murmur; while the second sound is distinct and much sharper than at the pulmonary cartilage. . . . . *Post Mortem* (twenty days later): heart, with 1½ inches of great vessels, weighs 13½ oz.; tricuspid orifice measures 4⅞ inches; pulmonary orifice measures 3⅛ inches; the tricuspid valves looked insufficient to fill the widened orifice, but no experiment done; left auricle very hypertrophous, in some parts wall = ¼ inch in thickness, its endocardium creaks in cutting: mitral orifice just admits end of index-finger, measures 1⅞ inches; edge is rugose, and valve is directed in funnel shape towards the ventricle; cordæ tendineæ very thick, are so shortened that practically the papillary muscles seem inserted directly into the valves; aortic orifice, water passes gradually through from above (but the coronary arteries were not tied); fine warty yellow vegetations especially on corpora aurantii; width of orifice = 2⅜ inches." . . . . (U. C. H., Females, vol. v., p. 292-299, Dec. 1850.)

Here the second sound was clearly defined at the left apex, there being no constrictive murmur; but that sound may be audible there, even when mitral constrictive murmur actually exists. On the other hand, so frequent is the association of regurgitation, that it is very rare to find a pure first sound at that point. At the mid-sternal base the first and second sounds are of natural character, except that they are sometimes feeble, from the smallness of the current sent on from the left ventricle. At the aortic cartilage, both sounds are feeble; at the pulmonary, both, on the contrary, and especially the second, are full and accentuated: if there be co-existent insufficiency of the tricuspid valve, this accentuation may, however, as in the case just referred to, be prevented from occurring.

1395. *Indirect Physical signs.*—Left auricular præ-systolic impulse, and right ventricular impulse, are frequently found as co-existences, in consequence of dilated hypertrophy being gradually produced in those situations. The pathological reasons of these enlargements are sufficiently clear. The area of percussion-dulness, especially to the right side, will on similar grounds be increased.

1396. *Pulse and Capillary systems.*—The character of the pulse and the pulmonary symptoms are the same as in regurgitant disease of this orifice. When constriction and regurgitation co-exist, as is commonly the case where there is constriction, either the murmur of regurgitation is heard alone, or a double to-and-fro murmur is distinguished; its two portions differing in quality and pitch.

TRICUSPID REGURGITATION.

1397. *Direct Physical signs.*—I have never known tricuspid regurgitation productive of thrill; the characters of its attendant murmur, which, for reasons there assigned, is often absent, are set down in a previous place [446]. The second sound is weak at the right apex, almost to extinction sometimes,—in all probability in consequence of the smallness of the current sent on through the pulmonary artery. Both sounds at the base and at the aortic cartilage may retain their natural characters; if the disease be pure, the second sound is weak at the pulmonary cartilage.

1398. *Indirect Physical signs.*—Commonly coincident with dilatation, simple or hypertrophous, of the right ventricle, the signs of these conditions are discernible:—Epigastric and right sternal impulse, of variable force, but out of proportion with that on the left side, unless the left ventricle be accidentally hypertrophous also, and increased area of percussion-dulness, mainly to the right. Hypertrophy of the right ventricle will prevent the enfeebling of the second sound appertaining to the pure regurgitation.

1399. *Pulse.*—The arteries, the pulmonary, which tricuspid insufficiency may be supposed to affect directly, cannot be felt; and there is nothing peculiar in the pulse of those derived from the aorta. The cervical veins distended, knotty, pulsatile, and refilling readily from below, may be the seat of thrill. But all these various signs are only found prominently in well-marked cases; if the regurgitation be slight, or if the right ventricle be very weak, venous pulsation, and thrill especially, may be altogether absent.

1400. *Effects on the Capillary systems.*—The pulmonary capillaries escape congestion in this form of disease. That the systemic capillaries, on the other hand, are essential sufferers, has been ably demonstrated by Dr. Blakiston: however, as I have already stated [1326], the sequence is not an absolutely necessary one; and dilatation of the cavities must, independently of regurgitation through any orifice, be admitted to play a most important part in inducing systemic stagnation. For the effects of this systemic stagnation, I would refer to the section on Dilatation.

Dr. Blakiston's results tend powerfully to show that obstruction of the cerebral capillary vessels and apoplexy are much more frequent in cases of heart-disease with, than without, stagnation of the systemic capillary circulation: hence the inference, that cerebral obstruction and apoplexy are more connected with the heart through dilatation and tricuspid regurgitation than through hypertrophy of the left ventricle.

1401. It may on first thought appear strange that such serious consequences should be ascribed to tricuspid regurgitation,—a phenomenon which the surmise of John Hunter, the arguments of Mr. Adams, and the experiments of Mr. King, would lead us to suppose exists in health, as a normal provision against overloading of the pulmonary capillaries.

But does such normal regurgitation really exist? Dr. Hope conceived that the absence of tricuspid regurgitant murmur in healthy persons sufficiently disproved the possibility of any such regurgitation: his argument was, however, valueless, for, as is well known, highly-marked morbid regurgitation even may exist without any murmur. Still I agree with Dr. Hope in his refusal to admit the reality of regurgitation in health: my grounds are, that if regurgitation existed, it would visibly affect the venous circulation in the neck, and that the experiments on which the doctrine is founded do not represent what common sense leads us to suppose must be the state of action at the tricuspid orifice during life. Besides, if, as is the fact, the valve, provided itself and the orifice be perfectly natural, will, after death, support water poured into the ventricle from the apex, how can we admit that during life the mechanical action shall be less complete? Conceding, however, *argumenti gratiâ*, that slight and occasional regurgitation is a normal fact, it might nevertheless be true, as actually is the case, that highly marked and permanent regurgitation should act as the source of most serious disorder.

TRICUSPID OBSTRUCTION.

1402. *Direct Physical signs.*—Constriction of the tricuspid orifice would theoretically give a diastolic murmur of maximum force at the ensiform cartilage. But this condition of the orifice is excessively rare, because atheroma and calcification are themselves extremely uncommon in this situation; and an amount of coarctation reducing the orifice to the size of the middle finger may not, as a case observed by Dr. Hope proves, give rise to murmur,—probably on account of the weakness of the current.

The following extract from my case-books seems to furnish the characters of tricuspid obstruction and regurgitation with preciseness, but there was no post-mortem examination to settle the point decisively.

"Wm. Hodson, ætat. 48; anasarca; constant cough, with rather profuse muco-purulent expectoration; no albumen in urine. Heart's apex beats feebly in fifth space; its dulness does not reach more than an inch to the right of the sternum. Both external jugulars considerably swollen, the right highly pulsatile from below, the left less so, though a small communicating vein, crossing the clavicle, pulsates slightly. At mitral apex, no murmur; first sound feeble, second sound clear, full and accentuated. At tricuspid apex, that is precisely at point of left costal angle [12], there is a soft double murmur, the diastolic division much better marked about level of the fourth interspace. At the mid-sternal base, just below the level of the third cartilages, it cannot be said that there is any murmur. The second sound at the tricuspid apex is frequently reduplicate, very rarely so at the mid-sternal base: the diastolic-timed murmur is louder and more prolonged than the systolic. Pulse visible in posterior tibials and radials, not in femorals. No cervical hum. . . . . In sitting posture second sound almost constantly reduplicate at tricuspid apex, whereas in at least twenty beats only once reduplicate at base, and not once at mitral apex (U. C. H., Males, vol. ix., p. 64-67).

The heart not having been lowered in position, it would appear unjustifiable to refer the diastolic murmur heard at the left costal angle to aortic regurgitation.

AORTIC OBSTRUCTION.

1403. *Direct Physical signs.*—In well-marked cases, systolic basic thrill may be caught; and the murmur already described [447] exists. The natural first sound of the heart is audible at the left apex, though slightly covered by the basic murmur: the second sound, weak at the apex, at the base, and at the aortic cartilage, may be slightly murmurish at the two latter points, in consequence of some regurgitant tendency. The second sound is occasionally reduplicate at the base. The systolic murmur may be prolonged enough to cover the second sound completely at the base; the latter may then, sometimes at least, be caught at the apex.

The murmur of chronic aortic constriction is one of those open to the greatest number of imitations. Of acute endocarditis, pressure by pericardial fluid or false membrane, as causes of similar murmur, I have already spoken; and, further, the distinction of this murmur from that produced by disease of the aorta close to the valves, and by abnormal communications within the heart, is far from easy. It has already been shown that displacement or twisting of the heart on its axis does not necessarily produce murmur, though this sometimes occurs. Of the blood-changes, producing systolic basic murmur, spanæmia is the most important; its distinctive characters have already been inquired into.

Weakness of action of the heart, or extreme smoothness of the constricted orifice, may prevent the development of murmur.

1404. *Indirect Physical signs.*—The impulse and altered percussion-sound of hypertrophy, with or without dilatation, of the left ventricle, are the common coincident signs. It sometimes happens that the murmur of mitral insufficiency is produced by the dilatation of that orifice following on dilatation of the ventricle.

1405. *Pulse.*—The pulse, in cases of moderate coarctation, is not materially affected; if the constriction be great, the pulse, though regular in force and rhythm, is small, hard, rigid, concentrated; hardness and force signify hypertrophy behind the narrowed orifice.

1406. *Effects on the Capillary systems.*—Aortic constriction exercises no direct effect on the pulmonary capillaries; some indirect tendency to stagnation arises from the difficulty experienced by the blood, flowing from the pulmonary veins, in its entrance into a ventricle, which, again, has itself great difficulty in discharging its contents.

It is truly remarkable to what an extent this coarctation may be carried, without producing systemic stagnation: the opening may be no larger than a pea, without leading to the very slightest œdema, even of the ankles. This immunity, however, only holds as long as the capacity of the ventricles and the width of the tricuspid orifice remain unaffected; if the blood becomes spanæmic, too, anasarca occurs independently of these latter changes. It has been conjectured that the peculiarity depends on the slowness of the circulation in old people; but the circulation is not always slow in old people, and young adults, with constriction of the aortic orifice, also remain free from systemic dropsy.

It seems theoretically probable that a constricted aortic orifice will weaken the effect of a hypertrophous ventricle on the brain.

AORTIC REGURGITATION.

1407. *Direct Physical signs.*—Diastolic basic thrill, though it has not fallen under my notice, may conceivably be perceptible, especially if the blood be at all spanæmic. I refer to a previous place [451] for the characters of the murmur of aortic insufficiency. The state of the heart's sounds is as follows:—The first, at the base, may be natural or very nearly so, dull, obscured by a soft murmur, or masked totally by a harsh one. The second sound, at the left apex, may be null, faint, or distinct and sharp; in the latter case, the sound heard is either that of the pulmonary valves transmitted, or is produced by fall of the aortic blood, during regurgitation, into the ventricle below [403]. At the aortic cartilage the first sound is of variable character; the second, a murmur, more or less marked. Both sounds may be perfectly natural at the pulmonary cartilage, and occasionally the second accentuated, though there be no mitral regurgitation.

Aortic regurgitant murmur is, as far as I know, constant, where its physical cause exists; weakness of ventricular action obviously cannot have the same effect in rendering a regurgitant, as a direct, murmur obscure. If a double aortic murmur exist, the systolic portion is best transmitted upwards in the course of the vessel, the diastolic generally downwards in the line of the sternum,—the latter peculiarity probably owing to the direction of the current and the aspirated quality of the murmur. The infrequency of direct tricuspid murmur and the coexistence of other signs of aortic regurgitation, will commonly prevent any error arising from the prominent character of the aortic murmur at the ensiform cartilage [1402]. It is conceivable that pressure on the outer orifice of the aorta just above the valves, might interfere with the closure of these, and so produce regurgitant murmur; in a case of displacement of the heart by pleuritic effusion, diastolic murmur existed under circumstances tending to connect the murmur with the displacement [664]. I have never met with a positive example of aortic *blood-murmur* diastolic in time, but such a murmur may be simulated in the following way:—spanæmia exists, with a strong systolic basic murmur; at the same time, deep-seated hum is present in the pulmonary veins; this hum is covered during the systole by the strong, blowing aortic murmur, but becomes audible during the diastole, when there is no aortic murmur to interfere with it.

Is murmur ever wanting in cases of aortic insufficiency? It may certainly be masked. Thus, in a case of aortic constriction with systolic basic murmur, and with superficial pulses visible and in the highest degree, the systolic murmur was so prolonged as to cover completely any diastolic murmur that may have really existed: but the diagnosis of aortic insufficiency, made on the ground of the extreme visibleness of the pulses, was proved to be perfectly correct at the post-mortem examination. I can conceive complete destruction of the aortic valves, with smooth surface, to exist without murmur; but have never observed the fact.

1408. *Indirect Physical signs.*—The concomitant signs are those of hypertrophy, eccentric or simple, of the left ventricle; frequently of aortic constriction, and occasionally of mitral regurgitation.

1409. *Pulse.*—The pulse is sudden, abrupt, short, jerking—a sudden fall back following instantly the rise of the vessel. Commonly regular in rhythm, sometimes notably lagging behind the systolic sound of the heart [422], occasionally bisferiens, each pulsation is, in rare instances, attended with thrill. Of visibleness and locomotion of the superficial pulses, enough has already been said [482].

1410. *Effects on the Capillary systems.*—Aortic regurgitation affects the pulmonary circulation, either indirectly through mitral regurgitation (of which it is itself the original cause), or more directly through the embarrassment produced by the collision of blood, falling from the aorta, with blood coming forward from the auricle. The tendency to sequential hypertrophy of the left ventricle, which arises mainly, I think, about the apex in these cases, does not, as in aortic constriction, afford any help to the circulation; on the contrary, by increasing the amount of distension of the aorta at each ventricular systole, it intensifies the force of the succeeding recoil.

The cerebral capillaries can only be affected secondarily through the pulmonary class; and the systemic capillaries are much in the same position. Regurgitation may exist to the highest amount without a particle of œdema of the extremities.*

PULMONARY OBSTRUCTION.

1411. *Direct Physical signs.*—Constriction of the pulmonary orifice exists with some frequency in cases of cyanosis, but coupled with such other conditions as render the attempt to fix on its special signs and effects difficult almost to impossibility. On the other hand, isolated constriction of the pulmonary orifice is necessarily rare, in consequence of the rarity of atheromatous and other changes in its valves. The characters of the attendant murmur will be found elsewhere [448]. Dr. Hope teaches that a pulmonary constrictive murmur seems closer to the ear than an aortic, and on a "higher key, ranging from the sound of a whispered *r* towards that of an *s;*" the reason being that the pulmonary artery is nearer to the surface of the chest than the aorta. As regards closeness to the ear, his statement is, doubtless, correct; as regards higher pitch, the reason assigned for this is obviously erroneous; and, indeed, Dr. Hope himself unconsciously admits this, by adding that he has known the murmur "fall below *r*, when the circulation was feeble and slow and the obstruction slight." †

1412. *Indirect effects.*—It is difficult to conceive that pulmonary constriction can exist to any extent without inducing dilatation, with more or less hypertrophy of the right ventricle, and systemic venous obstruction of a mechanical character. The pulse will probably present no uniform character.

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* *E. g.* E. Barnes, U. C. H., Males, vol. v., p. 216; but the fact is a common one: I have known this absence of dropsy, where the signs of regurgitation had existed for, at least, three years.

† Of three cases, given by Hope, in illustration of the murmurs of pulmonary constriction and regurgitation, one only terminated fatally, and here (Op. cit. p. 598) the notes only state "there was a murmur over the semilunar valves." Hence his volume gives no positive information on the subject. That a systolic murmur in an anæmic woman, is louder at the pulmonary than the aortic cartilage, does not by any means prove, as he appears to think it does, that the orifice of the aorta is not the seat of the murmur.

PULMONARY REGURGITATION.

1413. The theoretical characters of the murmur, significant of this state, have been already given [452]. No jerking character of the pulse, nor visibleness of the superficial pulses of the limbs would accompany a basic diastolic murmur of this origin. By a singular fatality, while a certain number of examples of such destructive disease or insufficiency of the valves, as must have led to full regurgitation, have been observed *post mortem* in this country, in but one, that I know of, have the physical signs been clinically established. Theoretically, the effects on the systemic and cerebral capillary circulation must be most serious; and a sensation of dyspnœa, arising from the smallness of the quantity of blood actually reaching the lungs by each systole, might, unless the force of habit would counteract this influence, be expected.

1414. The case referred to, observed by Dr. Gordon, is to be found in Dr. Stokes' recent volume. A boy, aged twelve, admitted into hospital with cold surface, congested feeble palpitation, very feeble pulse, cough, copious expectoration and moist rhonchus, had thrill over the entire cardiac region, and a double murmur, loudest at the base, and inaudible at the apex, without murmur, fremitus or visible pulsation of the superficial arteries. The thrill and "anomalous circumstances of the case" led Dr. Gordon to diagnose open foramen ovale: he was right, — a large opening existed between the auricles. But the pulmonary valves, thickened and shortened, allowed a column of water to pass through; the other valves were generally healthy.

Unfortunately the rhythm of the thrill is not mentioned. It would be opposed to our experience of the parallel aortic affection, to suppose thrill produced by pulmonary regurgitation; it probably depended on the obstructive influence of the diseased pulmonary valves, and would have been systolic in time.

1415. A loud musical diastolic murmur (where and in what direction most distinctly audible is not mentioned) was noted by Dr. Stokes, in a case of incompetence of the pulmonary valves, caused by widening of the orifice, the valves being sound: there was no aortic insufficiency.

1416. *General Diagnosis.*—In fixing on the seat of production of any given murmur, the first point for the observer to establish is the position of the heart itself: if this be abnormal, as it very often is in consequence of changes in its substance, preceding, coadvancing with, or sequential to the existing valvular disease, allowance must be made for the influence of such displacement in altering the maximum position of murmurs. Now, the general tendency is to some form of enlargement of the organ, and as enlargements lower it, the maxima points of murmurs are very commonly slightly lower than the maxima points of the corresponding sounds in health. Certain conditions of the aorta or pulmonary artery, of the lungs, pleura, or mediastinum, also throw murmurs into unnatural sites, by changing the position of the heart. Pleural fluid accumulations have, for an obvious reason [407], more influence in changing the audible point of apex, than of basic, murmurs.

1417. The state of the heart's substance also may exercise a direct influence on the intensity of murmurs. Thus hypertrophous texture, lying in the course of the circulation, behind a murmur, will intensify this, if it be direct; exercise little effect on it, if it be regurgitant: take the instance of an hypertrophous left ventricle with severally a constrictive and a regurgitant aortic murmur. Hypertrophous texture, lying in front of a murmur, is without effect upon this, if it be direct; intensifies it, if it be regurgitant: take the instance of an hypertrophous left ventricle with severally a constrictive and a regurgitant mitral murmur. Dilatation and weakening affections of the heart's substance weaken any direct murmurs through orifices on which such substance may play. Dilatation lessens the force of a regurgitant murmur, in so far as power of backward propulsion is concerned; but as, on the other hand, it allows of considerable accumulation of blood for regurgitation, the weakening influence is somewhat counterbalanced.

1418. The physical conditions of a given murmur positively existing at a given orifice, the character of that murmur may be seriously affected by the anatomical state of other orifices. Thus, aortic constriction may be almost murmurless, if there be any great co-existent mitral regurgitation, the aortic current is so seriously weakened by such regurgitation.

1419. Of two murmurs produced synchronously at different orifices, one may mask or cover the other. An aortic constrictive and a mitral regurgitant murmur may exercise this influence on each other; and a mitral regurgitant may completely drown a tricuspid regurgitant. In the interspace between the maxima points of two synchronous murmurs, however, a spot may generally be caught where the intensity of murmur is less than at either, and the quality different.

1420. An hypertrophous and widened state of the aorta will intensify its own regurgitant murmur; and if slight pouching exist at the sinuses of Valsalva, the rippling direction thereby given to the current will intensify both a direct and a regurgitant murmur. The state of the blood, too, is important: spanæmia greatly strengthens the force of murmurs of all kinds. The pulmonary veins and superior cava may be the seat of spanæmic murmur, simulating cardiac murmurs, especially systolic at the apex and diastolic at the base.

1421. Again, modified conducting power of adjoining textures may lead to error: thus an aortic regurgitant murmur may be heard better to the left than the right of the sternum, if the left lung be solid and the right emphysematous. Lastly, excessive irregularity and frequency of the heart's contractions, may make it impossible to determine the synchronism of a murmur.

1422. It follows from these facts that the diagnosis of valvular disease, though in the majority of cases sufficiently easy, must prove occasionally beset with various and serious difficulties,—difficulties in certain instances so numerous and so singularly combined, that it would savour more of temerity, than of experienced judgment, to venture on a positive opinion. But such cases are of very rare occurrence.

1423. *Symptoms.*—The more important symptoms of each variety of valvular disease are to be directly gathered from the statements made as to the influence of each on the different capillary systems. There are a few which may be considered common, though still not in precisely similar amounts, to all of the class. Wasting of strength, comparatively little of flesh, and sudden starts from sleep, which is habitually disturbed by frightful dreams, are very generally observed. Pain in the cardiac region is sometimes present, varying in amount from a slight aching sensation to the intensity of angina. It has been suggested that atheromatous and calcified conditions of the valves and orifices, interfering with the facility of stretching of these parts, may explain this. Palpitation is a symptom common, except in rare instances, to all valvular diseases carried to any amount. An albuminous state of the urine I have found most connected with tricuspid regurgitation; as also engorgement and œdema of the female pudenda and passive leucorrhœa.

1424. *Prognosis.*—The mean duration of the various valvular diseases is quite unknown: even the most serious are not incompatible with prolonged life, though sudden death, by syncope or coma, may occur during the course of any one of the number. In respect of comparative severity and danger, the chief valvular derangements may be placed in the following descending series:—tricuspid regurgitation; mitral regurgitation and constriction;* aortic regurgitation; and, least serious of all, aortic constriction. Dilatation of the heart always renders any valvular condition more dangerous: and, as a rule, hypertrophy, though to a less degree, has the same kind of effect. Hypertrophy of the left ventricle, however, and directly as its purity, may mitigate the effects of aortic constriction. I do not know from experience the influence of such hypertrophy on regurgitant disease of the aorta, and theory speaks in both ways.

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* Hence the danger of valvular diseases, as a class, is not to be estimated by the amount of murmur they habitually entail,—for tricuspid regurgitation and mitral constriction, which hold so high places in the scale of fatality, are

1425. *Treatment.*—Whatever be the mechanical condition of diseased valves, and whatever be the nature of the diseased action, inflammatory, fibrin-depositing, fatty, calcifying, that has impaired their freedom of function, they lie themselves without the pale of direct treatment. Valvular disease once chronically established cannot be cured: we can neither remove deep-seated induration-matter, atheroma, nor calcareous substance, nor lengthen tendinous cords that have been shortened by morbid processes. Hence the importance of at once guarding as much as possible against one of the frequent causes of acute endocarditis—namely, rheumatic fever, and of preparing for the active treatment of that inflammation the moment there is reason to suspect its immediate advent. Of the prophylaxis of non-inflammatory valvular changes, nothing can at the present day be said.

1426. But though these diseases be anatomically incurable, their worst functional effects may be long, in some cases indefinitely, averted, by measures accordant with a common-sense view of their nature and ratified by experience. Whatever be the valve implicated, the treatment is directed not towards its own disease, but towards the moderation or prevention of

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precisely the two with least constant murmur. This is sufficient proof that implicit faith—nay, any faith—in respect of prognosis, must not be put in the conditions of valvular murmurs; and it may be well here, again, to remind the younger reader of what may be fully gathered from the description in the First Part, that there is no direct connection between the amount of disease at an orifice and the intensity of an existing murmur;—the very *weakness of a murmur may, indeed, be a fatal sign.*

hypertrophy of the muscular substance of the heart or dilatation of the cavities,—the control of extra strength or the support of undue weakness,—and the removal or relief of any symptoms that may arise. Practically the treatment of valvular diseases comes to be that of hypertrophy or dilatation, due regard being had to the influence exercised by the mechanical obstruction on the character and tendencies of those conditions of the heart's substance. Thus, if with valvular disease there co-exist dilated hypertrophy, occasional very moderate venesection, or the abstraction of blood by cupping or leeching over the præcordial region, will be advisable: more so in cases of superadded mitral regurgitation than of aortic constriction, for reasons easily inferrible from the influence of such hypertrophy in these two valvular diseases. Profuse blood-letting is, under all circumstances, absolutely improper: it cannot remove either the disease of the valves or of the heart's substance, and may induce anæmia, excitement of the heart, and early dropsy. If dilatation exist, with or without attenuation, venesection is decidedly contra-indicated: three or four leeches may, however, even under these circumstances, be applied to the cardiac region for the relief of palpitation, anginal feelings, or sudden engorgement of the right cavities. Dry-cupping and mustard poultices should, however, first be tried: they often give quite as much relief, and without any concomitant sacrifice.

1427. Of the value of *diuretics*, when dropsy has appeared, no doubt can be entertained: but long before matters have proceeded to this point, medicines of the class seem useful, by diminishing the quantity, without impoverishing the quality, of the circulating fluid, and so lessening the tendency to clogging of the intra-cardiac circulation and the proneness to palpitation. The acetate, nitrate, iodide, and bitartrate of potass (the latter in two drachm doses largely diluted), nitric ether, compound tincture of iodine, the infusion and spirits of juniper, the decoction of chimaphila, and other agents of the class may be variously combined and successively employed. The action of these diuretics is facilitated by occasional small doses, at bedtime, of blue pill and squill. When the kidneys are much congested, removal of that congestion must be effected, wholly or partially, before the organs will act. Cupping to a small extent or dry-cupping in the renal regions, sometimes exercises a remarkable effect in facilitating the action of diuretics by removing congestion : in the same way is to be explained the favourable influence of mustard poultices and even *blisters* to the loins. Urine, more or less impregnated with albumen, previous to these measures, becomes perfectly free, to ordinary tests, of that principle after their employment.

1428. Regularity of alvine discharge, in all diseases important, is particularly essential in these cases, to prevent engorgement of the liver, and obviate the necessity for effort in the act of defecation. Continued purgation is, however, to be avoided prior to the occurrence of dropsy.

1429. If at any time the stomach be loaded, and its replete state excite palpitation, an *emetic* seems clearly indicated : the sulphate of zinc is the fittest. But where the circulation is very much embarrassed, emetics sometimes increase considerably that embarrassment, and unhappily it is far from easy to fix beforehand the probable influence of an emetic in any particular case. If there be doubt, it is better to refrain and allow the stomach to free itself gradually in the natural manner. Dr. Hope was of opinion, and with much *à priori* reason in his favour, that a state of prolonged nausea, by causing languor of the circulation, promoted the formation of fibrinous coagula within the heart.

1430. Where the lungs tend to engorgement, especially in mitral disease, *expectorants*, either ipecacuanha, lobelia, stramonium, or squill and ammoniacum or senega, according to the active or passive character of the symptoms, become necessary. Dry cupping of the chest, sinapisms, and blisters materially relieve such engorgement. *Anti-spasmodics*, chloric ether, ammonia, &c., are necessary in paroxysms of dyspnœa. *Opiates* cannot be dispensed with at night in advanced cases ; but where the valvular obstruction is considerable and the heart weak, caution is required in their exhibition. *Antacids*, carminatives, and light bitters, and other stomachic medicines, relieve the gastric discomfort so common in the victims of valvular disease; but the latter must not be permitted to an extent to stimulate the appetite to any great amount.

1431. The propriety of administering mineral *tonics* will, in the main, turn on the condition of the heart's substance,—whether dilated or hypertrophous,—a subject already considered. Theoretically, invigorating tonics are more advisable in aortic constriction than in mitral regurgitation. Anæmia peremptorily calls for iron.

1432. *Issues* or *setons* to the præcordial region sometimes relieve local pain and discomfort; they are otherwise valueless. At the very earliest period of chronic inflammatory changes in the valves, it is possible that ioduretted frictions may promote absorptive action.

1433. The treatment of dropsy has already been described.

1434. In regard of diet, no constantly applicable rules can be laid down, except that moderation is important both in solids and, especially, in fluids. Exercise should never be pushed to fatigue, and laborious efforts of all kinds systematically avoided. When the dyspnœa is of serious character, the effort of mounting a flight or two of stairs, especially if the patient be obese, is more than he dares encounter: his instinct apprises him of the imminent risk of the attempt. Under these circumstances the common plan is to recommend the patient to occupy the ground floor,—a mode of living having its own hygienic disadvantages, unless in houses constructed on an unusually large and commodious scale. My friend, Mr. W. B. Hutchinson, has, in a case of this kind, succeeded in enabling the sufferer to use a sleeping-room at the top of the house, much to the improvement of his general health, by having him raised in a chair suspended by solid pulleys to the ceiling of the staircase,—something on the plan of the well-known contrivance at the Coliseum.

1435. I have not spoken of digitalis. The action of this medicine, when it really does slacken the circulation materially, is rarely demonstrably beneficial, sometimes seriously mischievous, always hazardous. The obstruction to the circulation through the heart, necessarily produced by valvular disease, tends to promote coagulation of the blood within it; and on the evils of that coagulation it is needless to insist. If digitalis be employed at all, it is least dangerous in its diuretic form, that of infusion.

MALPOSITIONS.

1436. I. Malpositions of the heart are *congenital* or *acquired.* Of the *congenital* class the extra-thoracic, cephalic, and abdominal ectopiæ are totally without clinical interest. Not so, congenital malposition of the heart in the right half of the thorax; for this being a kind of displacement producible at all periods of extra-uterine life by a variety of diseases, it becomes of importance to have a means of positively distinguishing the malposition of congenital origin. Now this means is furnished by the position of the liver and spleen; for, except in very rare instances, when the heart has formed and grown on the wrong side of the spine, the abdominal viscera, also, have been transposed. Where this guide is wanting, the distinction may be very difficult; and the observer is only justified in pronouncing the mal-position of the heart to be congenital after he has succeeded in excluding every possible morbid source of acquired displacement.

1437. II. The diagnosis of *acquired* displacements is to be made by the position of the apex-beat and of the impulse of the organ generally,—also by the comparative intensity of the sounds in different parts of the chest.

The varieties practically important, namely, Detrusion, Elevation, and Procidentia, are described, and their modes of production illustrated in a former place [302—306]. The clinical interest of them all depends upon the light thrown by their discovery on the diagnosis of various intra-thoracic diseases. They themselves produce no symptoms; and the restoration of the organ to its natural position, can of course only be effected by removal of the condition displacing it.

CYANOSIS.

1438. Cyanosis, morbus cæruleus or blue disease, are the names applied to a symptomatic state, prominently characterised by blue discoloration of the tegumentary membranes, and known by experience to be connected with various malformations of the heart and perverted modes of origin of its great vessels. The general tendency of the more common of these malformations is to alter the relationships naturally subsisting between the two sides of the heart and the two kinds of blood—dark and florid. But some of the number act in other ways, and the entire series may be referred, as species, to three classes, in the following manner:—

A. *Conditions causing direct communication of the arterial and venous circulations.* (*a*) *In the heart.*—Open foramen ovale; deficiency of part of the ventricular septum; perforation, of ulcerative or other character, throwing the auricles or the ventricles or all the four cavities into, practically speaking, a single cavity; heart formed of one auricle and one ventricle, the latter giving off one artery, which divides into a pulmonary artery and aorta, &c. (*b*) *In the great vessels.*—Freely pervious ductus arteriosus; aorta rising from both ventricles, or from the right ventricle, or from a trunk common to itself and the pulmonary artery, &c.

B. *Conditions causing distribution of black blood almost solely to the systemic capillaries, and of red blood to the pulmonary capillaries, without, practically speaking, any intermixture of the two kinds of blood.* Here appear cases where the aorta rises from the right, and the pulmonary artery from the left, ventricle,—the venæ cavæ, as in the natural state, communicating with the right, and the pulmonary veins with the left, auricle;—where, consequently, there are two distinct circulations, communicating alone by the ductus arteriosus, if this remain open.

C. *Conditions obstructing the entry of blood into the lungs, or intensely congesting them, so as to prevent oxygenation.* (*a*) *In the heart*:—Excessive smallness of the right ventricle; extreme narrowness of the tricuspid orifice: these states disturb the process of distribution of blood *to* the lungs. Great contraction of the cavity of the left ventricle, great coarctation of the mitral orifice: these conditions prevent the return of blood *from* the lungs. (*b*) *In the great vessels*:—Partial or complete obstruction of the orifice of the pulmonary artery.

1439. *Symptoms.*—The most striking symptom of Cyanosis is blue, leaden, purplish, violet, or almost black discoloration of the skin; the discoloration may pervade the entire surface of the body, but it is always most marked at the internal canthi, lips, nose, ears, below the eyes, and at the tips of the fingers and toes. Changeable in intensity from time to time, it is greatly deepened by palpitation, emotion, dyspnœa, and all conditions throwing any additional obstacle in the way of the circulation; generally speaking, the tint lightens materially after death. The ends of the fingers, if life continue for any length of time, grow more or less bulbous; in adults this peculiarity of form is sometimes singularly well-marked; at the same time the nails are incurvated; and the patients are said to be particularly subject to whitlow. The temperature of the surface and mucous canals, at their orifices, is low; the patients are constantly chilly, and sensitive to the least fall in the thermometer. Œdema of the feet sometimes exists; the frame is weak, the muscular and adipose systems ill-nourished. Nevertheless, the viscera may be of full size;* their colour does not seriously differ from that of health, — but this may in great measure depend on their tint, as well as that of the skin, changing after death.

Though rarely the subjects of serious habitual dyspnœa, cyanotic persons frequently suffer from paroxysmal difficulty of breathing—often the result of disturbed circulation caused by fits of petulance and passion, to which they are, above the average, prone. Cough, either dry or attended with watery expectoration, probably through œdema of the lungs, frequent attacks of congestive bronchitis, added to, sometimes, the physical signs of emphysema, constitute the sum of pulmonary symptoms and morbid states. Palpitation active, tumultuous, with strong diffused impulse, a pulse ranging from 120 to 160, irregular in force and rhythm, often coexists with the dyspnœal paroxysms above referred to; syncopal tendency and actual syncope, though rare, do sometimes actually occur; the eyes are prominent, the expression wild, and the arms are tossed violently about. There is no particular pulsation observable in, even, the most deeply-coloured parts. Convulsions, somnolence, and semi-coma occasionally occur; and reflex phenomena, such as jerking action of the limbs and grinding of the teeth, are frequent during sleep.

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* Vide case of Cyanosis, Med. Chir. Trans., vol. xxv.

1440. *Physical signs.*—The physical signs in the cardiac region vary according to the anatomical condition. In the most intense form of cyanosis, where the whole system is fed with very little else than black blood, as in cases of transposition of the aorta and pulmonary artery, there need not of necessity be any abnormal physical sign at all. In the most common anatomical state (open foramen ovale and constriction of the orifice of the pulmonary artery, with sequential hypertrophy, simple or eccentric, of the right ventricle), systolic basic thrill, doubtless in the pulmonary artery, may be felt; the systolic murmur of pulmonary constriction may be traced up to the second left cartilage; and the signs of an hypertrophous state of the right ventricle will be coincidently found. I do not know that we are in a position to assert positively, that patency of the foramen ovale will in itself cause murmur; for in all records that I have met of the coexistence of the two things, either constriction of the pulmonary orifice is actually stated to have existed, or it may have existed—all mention of the vessel being omitted by the narrator. I once met with a systolic murmur in a cyanotic child, audible at a little distance from the surface; I saw the boy but once, and unfortunately neglected to note the topography, if I may use the expression, of the murmur.

1441. Cyanosis is not always congenital. Of seventy-one cases, forty only existed at birth.* When appearing for the first time in early childhood, possibly enlargement of the heart gradually opens out the foramen ovale, and so induces the intermixture of the two kinds of blood; or perhaps congenital constriction of the pulmonary artery increases. In the adult, the appearance of cyanosis has sometimes been traced to a blow, a fall, or an effort; possibly some forcible separation of the edges of the foramen ovale had occurred; sometimes ulcerative openings in the auricular or ventricular septum have proved the immediate cause. Occasionally the external evidences of the malformation have been but slight, until the accidental occurrence of serious bronchial or pulmonary inflammation.

1442. It has been theoretically said that cyanosis, like other states in which venosity of the blood predominates, "offers a complete protection" against tuberculisation. Several cases are on record showing the fallacy of this doctrine absolutely stated; † what degree of antagonism, if any, really exists between the affections, has never been submitted to clinical investigation.

Cyanosis seems to have some share of influence in producing pericarditis: but this point also requires further examination.

1443. *Diagnosis.*—The only affection in the new-born infant with which it appears possible to confound cyanosis, is apoplexia neonatorum (intense congestion, with extravasation of blood, of the membranes of the brain and spinal cord), characterised by great lividity of face, swollen scalp, feeble action of the heart, low, irregular respiration, clenching of the hands, convulsive ctions, torpor, chilliness—conditions lapsing into fatal asphyxia, nless treatment, especially bleeding from the umbilical cord, rove successful. But the tint of skin in cyanosis is different, luish, not livid; the scalp is not swollen; nor is there general umidness of the upper part of the body; the action of the eart is rather in excess than deficient in strength; and the espiration is not laboured, irregular, and slow.

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* Stillé; Amer. Journ. Med. Sc., N. S., viii.

† Fearnside, Association Journal, March, 1854.

1444. *Mechanism.*—It would be altogether beside the purpose f a work like the present to discuss the theory of cyanosis; but justification of the influence ascribed, in the classification bove given, to intermixture of venous with arterial blood, it ust be stated, that the doctrine of venous stagnation adopted y Morgagni and M. Louis, to the exclusion of ordinary ideas oncerning that intermixture, seems scarcely satisfactory. How that doctrine reconcilable with the fact, that the most intense enous obstruction may arise in the adult without inducing true yanotic discoloration? How comes it, too, if communication etween the two sides of the heart be so unimportant, that, in ve only of seventy-one cases of cyanosis collected by Stille was uch communication wanting? Is it not likely that two things, constantly found together, act as cause and effect; and that, here a widely open foramen ovale has been found, as it cer- inly occasionally has, without previous cyanosis, some corrective ondition, either organic or dynamic,* has existed, to prevent e intermixture? Doubtless constriction of the orifice of the ulmonary artery will increase the darkness of tint, by inducing enous stagnation; but I do not think there is evidence to show at, unassisted, such constriction can produce cyanosis.

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* If the pressure of the current on either side of the opening be equal, ere is no reason why each current should not pass on without commingling all, or more than very slightly, with the other. Such nicely-balanced essure must be rare, however, as the right auricle tends to become dilated d somewhat hypertrophous; and so, precisely, it is very rare that the foramen ale is open to any extent without intermixture of the two bloods, and con- quent cyanosis. It is probable also that a pretty free admixture is required.

1445. *Treatment.*—The treatment of a case of cyanosis resolves itself into the prevention, as far as possible, of paroxysms of dyspnœa and palpitation. Tranquillity of the circulation, by the avoidance of all emotional excitement, mental or bodily, and of all conditions likely to congest the lungs, the liver, and the abdominal organs, is to be aimed at; the temperature of the skin maintained by warm clothing, moderate exercise, and friction; and that of the body, generally, raised, if the stomach be not disordered thereby, by the free consumption of oil, fat, gum, and other aliments of respiration.

RUPTURE AND PERFORATION OF THE HEART.

COMPLETE RUPTURE.

1446. Rupture of the heart's substance into the pericardium, from all causes, spontaneous and other, indiscriminately, takes place with much greater frequency in the left ventricle than any other part of the organ:* but, if cases originating in external violence alone be considered, the right ventricle has, according to existing records, suffered more frequently than the left, in the proportion, according to Ollivier, of eight to three.

1447. Variable in size and form, sometimes smooth, sometimes ragged at the edges, the inner and outer openings—that is, the pericardial and the endocardial—may correspond or not; in the latter case, a sort of sinus exists in the wall of the ventricle, connecting the two. There is usually a single rupture only, but so many as five have been seen; sometimes a single opening on the inner surface of the ventricle communicates with two or more on its pericardial surface. The strata of muscular tissue next the endocardium, again, may be pretty extensively destroyed, while a tiny perforation only in the pericardium can be discovered. Although there is every reason to believe that, in the great majority of cases, rupture of the heart is perfectly sudden in its occurrence, it is clearly a gradual process in some rare cases; for instance, where hæmorrhagic softening of tissue has led to its occurrence: under all circumstances, the final breakage of the last few muscular fibres and outer serous membrane, that separate the pericardial and endocardial cavities, is instantaneous. The fissure runs very nearly three times as often parallel to, as at right angles with, the main fasciculi of the heart's fibres.

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* In 52 instances of rupture collected by Gluge (Path. Anat.) the left ventricle was the seat of the rupture in 37 cases; the right ventricle in 8; both ventricles in 2: the right auricle in 2: the left auricle in 3.

1448. Considerably more frequent in males than females, in the ratio of 36 to 16, rupture of the heart is favoured by advanced age: it becomes comparatively frequent after the fiftieth, still more so after the sixtieth year. Immediately induced by efforts of some kind, by fits of passion, by great and abrupt thoracic congestion, as through sudden immersion in the cold bath, by blows and other injuries to the præcordial region, the way is paved for its occurrence by various textural changes of the heart's substance or aorta: as fatty accumulation under the pericardium; intra-sarcolemmous fatty change; softening of undetermined kinds; dilatation with attenuation; local suppuration, ulceration, possibly gangrene; hypertrophy, with, probably, fatty change superadded; hydatids; calcification of the ruptured tissue; aneurism of the left ventricle; local destruction of the endocardium; apoplexy of the heart (the question has, however, been raised, whether in cases where blood-infiltration and rupture have been found together, the former may not have depended on gradual advance of the latter); coarctation of the arch of the aorta, and, as in the case of George I., aneurism of that vessel. In all probability, though old narratives say the contrary, the heart's texture is never perfectly sound; even where the cause of the rupture has been external violence, some alteration of tissue has been generally found by recent observers.

1449. *Symptoms.*—In the majority of cases, rupture of the heart, if itself complete, at all extensive, and instantaneous, kills instantaneously. The hand is suddenly carried to the front of the chest; a piercing shriek uttered; some convulsive twitches occur, and the patient expires: or sudden loss of consciousness, from which recovery never takes places, marks the event.

1450. There is a class of cases, too, in which from the plugging of the fissure by coagula, the amount of extravasation of blood into the pericardium is insufficient to stop the heart's action at once. Under these circumstances, a patient has been known to survive fourteen hours, with pallor, cardiac anguish, clammy sweats, coldness of the surface, feeble fluttering pulse, and sighing respiration; eventually going off quietly in a state of coma. The fact of death occuring so slowly suggests the question whether the permanent closure of a fissured opening does not fall within the range of the possible; and certainly one case of sudden death from rupture has been recorded, in which a former rupture was discovered, firmly filled by a fibrinous coagulum adherent to the wall of the heart.

The protraction of life for many hours after the probable moment of the perforation, is illustrated by the following case:—

William Hutchinson, ætat. 48, presenting all the signs and symptoms of general dilatation, with hypertrophy, of the heart (the organ weighed twenty-two ounces), mitral and tricuspid regurgitation, enlarged liver, pulmonary apoplexy and anasarca, had slightly improved in the majority of his symptoms during three weeks stay in hospital, when a change for the worse, attracting the immediate attention of the nurses, occurred on the morning of the 25th August: the countenance had become of pale yellowish earthy aspect; the conjunctivæ bloodless; the face and thorax covered with cold sweat; the respiration laboured and frequent; the pulse weaker and smaller than previously; uncountable from frequency; feet considerably œdematous and cold; no pain complained of; restlessness intense,—change of posture almost every minute; tongue particularly clean; bowels naturally open. During the night of the 25th, insomnia, constant restlessness; dressed himself and sat on side of bed greater part of the night, wishing to go home. August 26th. In the morning: does not know his children; body exhales an earthy, and somewhat putrescent odour; extremities cold; pulse almost imperceptible, uncountable from frequency; respiration more difficult and frequent than yesterday; cold sweats: so late as 3·30 p.m. this day he got out of bed to sit up; passed urine under him; at 5·0 p.m. hacking cough set in; shortly after, the respiration became unfrequent, occurring at such rare intervals, that each breath seemed to be his last. . . Sank at 5·59 p.m., without any change of symptom. On examination (made, in my absence, by Dr. Hare) about two ounces of dark blood, partly coagulated, partly fluid, were found on the surface of the heart and root of the aorta; after careful search, a minute aperture, barely admitting an ordinary sized pin, was letected close to the apex of the right ventricle, and appeared to communicate vith the interior of that cavity in an oblique direction downwards, and to the eft. (U. C. H., Males, vol. viii., pp. 334—354).

The slowness of dissolution here was, in all likelihood, due to he minute scale of the perforation, and the slowness with which he blood filtrated through it. The probability is, that perfora-ion was completed during the night of the 24th, or morning f the 25th; and that consequently the patient survived the ccurrence thirty hours on the lowest computation.

1451. A third class of cases exists, in which sudden cardiac nguish, attended with a sense of constriction, extreme dyspnœa, ain extending from the præcordial region to the left shoulder, oldness of the surface, giddiness and faintness, cramps, and mall irregular pulse, are observed,—the whole series undergoing emission, nay, actually, it is said, totally disappearing temporarily, again to return with greater intensity than before, and lose in death. In instances of this kind, in all probability, the eart's fibres have given way in successive layers.

1452. *Treatment.*—The greater number of cases of at all xtensive rupture have terminated fatally, before medical ttendance has been procured. Should the patient be still iving, when first seen, such treatment should be employed as common-sense view of the symptoms would suggest; for it is ore than probable that the precise nature of what has occurred ill rarely be diagnosticated with surety. Theoretically, the lear indication is to maintain the circulation in movement, with s little work on the part of the heart as possible; the head hould be placed low, sedatives and slight stimulants administered, the surface kept artificially warm, and the very slightest ovement of the body, as far as possible, prevented. If reaction ccurred, blood should be cautiously taken from the arm.

In instances of slow perforation, of which an illustration has been given, the nature of the occurrence is, in the first place, beyond the reach of sure diagnosis. In an individual, dying more or less rapidly of profound organic disease of the heart, the increased uneasiness and increased disturbance of the pulse, depending on perforation, might be taken for indications of polypoid formation within the cavities, or rupture of a portion of valve or chorda tendinea. Fortunately, however, the treatment will not err in consequence: in any one of these cases the immediate management is that fitted for nervous shock and cardiac obstruction.

PARTIAL RUPTURE.

1453. Partial ruptures of the heart, or of its valvular apparatuses, are not extremely unfrequent. Rupture of the papillary muscles, permitting the ruptured ends to float, as it were, free in the cavity of the ventricle; rupture of the substance of the mitral or tricuspid valves, or of their tendinous cords: and of the pulmonary and aortic valves,—have all been observed *post mortem*, and their symptoms occasionally noted with more or less precision during life.

1454. The immediate symptoms of the rupture of a chorda tendinea, especially if endocarditis have caused the accident, are not very prominent. The difficulty of the circulation through the heart, however, is increased; the pulse becomes very irregular, and symptoms precisely like those of the sudden formation of blood-concretions in the ventricles, make their appearance. The murmurs, obstructive and regurgitant, of the implicated valve ensue; but they may have pre-existed, as consequences of the endocarditis itself, and in that case positive diagnosis is an impossibility.

Sudden rupture of any number of chordæ tendineæ, or of a papillary muscle, especially when occurring independently of acute disease of the heart, produces very marked symptoms. Cardiac pain and anguish; palpitation irregular in force and rhythm; small, irregular, frequent pulse; syncopal tendency, overwhelming dyspnœa, dread of dissolution, pallor, coldness of surface, jactitation of the limbs,—all this suddenly occurring, in connection with a regurgitant systolic apex-murmur, which had been known not to have existed previously, might guide to the diagnosis, but not justify a positive assertion on the subject.

In a case of rupture of one of the aortic valves, observed by myself, symptoms such as those just described were attended with regurgitant basic murmur.

1455. The character of the symptoms points to the propriety of administering stimulants and sedatives: sinapisms or the turpentine fomentation to the cardiac region relieve the anguish and constriction felt in that situation.

RUPTURE OF A CORONARY ARTERY.

1456. An individual, subject to palpitation, had a sudden attack of retching, with cardiac anguish, and died in an hour. The left coronary artery, widened, hardened, and fragile, was broken across from the aorta; effusion of blood had taken place between the pericardium and great vessels.*

ANEURISM OF THE HEART.

1457. Aneurism of the heart occurs in two forms, correponding to the fusiform dilated aneurism, and the lateral imple sacculated aneurism, of arteries (*vide* Diagram III., figs. , 3, p. 737): that is, a general and tolerably equable dilatation of a portion of the wall of a ventricle exists, or a pouched ulness rises abruptly, with or without constricted orifice, from the ventricle. Of either kind, aneurism is almost peculiar to the left ventricle. The compound sacculated aneurism of arteries, with injury to their walls (Diagram III., fig. 4), is also imitated by the heart, when destruction of the endocardium and, more or less extensively, of the nearest strata of muscular fibres precedes the pouching process.

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* Lombard; Gaz. Méd. de Paris, iii. 644.

1458. In the majority of cases aneurism forms slowly, dependent, as it is, for existence on chronic changes in the substance of the heart; in some cases, however, its formation is an acute process, induced by ulceration or rupture of the endocardium and contiguous fibres.

1459. Greatly more frequent, as 3 to 1, in males than females—hitherto most commonly observed between the ages of twenty and thirty and in very advanced life, though few ages are actually exempt from the possibility of its occurrence,—having the ground-work laid for its formation in inflammation, fatty change, simple softening, or pseudo-fibrous infiltration of the heart's substance,—aneurism has been immediately traced, in a certain number of instances, to external injury, violent efforts, forced retention of the breath, and similar agencies, suddenly throwing an intense strain on the walls of the left ventricle. In the majority of cases its origin is slow and insidious—in fact, latent.

1460. *Symptoms.*—Under all circumstances the symptoms are obscure. When the onset is presumedly sudden, this has been announced by severe præcordial pain, orthopnœa, general agitation, dread of dissolution, syncopal tendency, and frequent, small, irregular, languid pulse. But obviously there is nothing distinctive in these symptoms: they indicate that the heart has received a deep shock of some kind, and nothing more.

On the other hand, where the disease is of slow origin and course, its symptoms do not seem to become serious, unless itself is carried to a great amount, or dilatation, with more or less hypertrophy is added. The effects of such dilatation are then developed—systemic stagnation and its attendant evils in more or less complete array. At all events, it will be admitted that past records do not supply the materials of a clinical history of aneurism, clearly distinct from that of affections of the ventricles and of the orifice, however complete an anatomical one they have been made to yield by the zeal of Mr. Thurnam. Every known symptom of cardiac disease has been present, it is true, in these cases; but, as there either were positively, or may possibly have been, other morbid states present (I refer to narratives which make no affirmation as to the absence of such states), capable of causing the symptoms in question, it would be perfectly unjustifiable to ascribe them to aneurism.

1461. *Physical Signs.*—And there is as great a dearth of knowledge of positive physical signs. The position of the heart's maximum impulse may perhaps be transferred from the apex to the base, where a sacculated aneurism springs from the neighbourhood of the base of the ventricle; probably, if the sac were prominent, the action of the heart would be attended with pericardial rub, not only systolic, but diastolic. Systolic murmur of blowing quality, of maximum force at the left apex, ias been observed, undistinguishable from the murmur of mitral egurgitation. Though seemingly probable, *à priori*, that a louble murmur might be produced by the ingress and egress of lood from the sac, where the orifice of this was narrow, xperience shows that the diastolic portion may be completely vanting.* The signs of dilated hypertrophy frequently, of alvular disease sometimes, are coincidently met with.

1462. Death may occur suddenly from rupture of the sac into he pericardium, or, through an adherent pericardium, into the leura: in the majority of instances the patient is slowly worn ut with the symptoms of dilatation.

1463. There is no special treatment for this affection: the ymptoms and signs guide the physician to the adoption of the easures best adapted for hypertrophy or dilatation: the effects f systemic stagnation are to be averted or removed by the plans lready described.

ANEURISM OF THE VALVES.

1464. Sacculation, pouching, or aneurism, of the valves has casionally been observed; always, as might be expected, in the direction of regurgitation, and produced by the force of the current playing on textures weakened in resistent power by chronic disease. This condition, anatomically known in the mitral, tricuspid, and aortic valves, has at present no clinical history.

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* *E. g.* In Case V. of Dr. H. Douglas's Collection of Cases of Heart-sease, Edin. Monthly Journal, 1850.

POLYPOID CONCRETIONS.

1465. Formation of fibrinous coagula within the heart during life is promoted by retardation of the circulation, occurring towards the close of exhausting diseases, in connection with weakness of the organ, or mechanical obstruction to the circulation in its interior. Hyperinosis carried to a great extent, as in pneumonia, has, on another principle, a somewhat similar tendency [778]. The condition of the blood in pyohæmia, and in other of the heteræmiæ likewise, favours coagulation within the heart; but endocarditis is, of all affections, its most frequent and effectual cause.

1466. The symptoms and treatment of coagulative accumulation of blood within the heart in endocarditis have already been described.* In chronic diseases with languid circulation, the risk of coagulation should be averted as far as possible by abstaining, at least in any quantity, from the use of nauseant medicines and digitalis, the obvious effect of which must be to slacken the circulation. Blood-letting should also be refrained from, unless under some urgent necessity. The influence of diluents, recommended for the purpose of rendering the blood less coagulable, can scarcely be trusted to,—and by producing a watery plethora they may do more harm than good. Small quantities of bicarbonate of potass, taken two or three times a day, seem to me the most promising prophylactic.

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* Continual nausea and vomiting have occasionally been observed for several days before death in persons, whose hearts contained coagula evidently formed during life: but the connection of the two things is not perfectly clear; concretions may certainly form without disturbing the stomach.

CHAPTER III.

DISEASES OF THE AORTA.

AORTIC PULSATION.

1467. Aortic pulsation, or abdominal or epigastric pulsation or palpitation, as it has been variously called, is a peculiar functional affection of the aorta, distinguished by more or less throbbing action of the vessel. Although very positively observable in the thoracic aorta, it is best known in the abdominal division of the vessel.

1468. *Symptoms.*—Pulsation at the epigastrium, more or less constant, but aggravated by various influences, such as brisk movement, nervous excitement, irritation of the bowels, or constipation and dyspeptic disturbance, is, as its title indicates, the prominent feature of the complaint. In rare instances, complete intermission of the throbbing action may occur,—a fact important in respect of diagnosis. When strongest, the pulsatile movement is attended with a feeling of sickness or faintness, or pseudo-globus; there is no actual pain experienced, except the adjacent parts be accidentally extra-sensitive. The epigastrium, however, always bears pressure worse than in healthy people, and may be extremely tender.

1469. *Physical Signs.*—In well-marked cases, pulsation may be easily seen at the epigastrium, especially in thin people; rarely at the umbilicus. The hand, laid on the surface in the course of the vessel, receives a forcible forward impulse, slapping rather than heaving, jerking, quick, abrupt, without distinctly expansile character, but bounding and free,—varying in degree from an action so trifling as to be scarcely perceptible, to one sufficiently powerful to shake the bed. As a rule, there is no lateral expansion to be felt; but, if hardened tissue lie on the confines of the vessel, such expansion may at the least be closely simulated. Unless the blood be anæmic, no thrill is to be caught. The vessel, if the patient be very thin, may be reached on either side by the fingers, and slightly moved laterally. The transverse limits of the vessel under percussion are natural; but it must be confessed this is a fact very difficult of establishment: the abdominal wall should first be steadily depressed for a minute or so, all gas and fæcal matters, as far as possible, pushed sidewards, and then the vessel carefully percussed. Aortic pulsation may exist in the highest degree without murmur,—a single systolic* impulsive sound being alone audible; or a systolic and a diastolic sound may be heard, the latter the fainter greatly of the two. Or, what is more common, a single systolic blowing murmur, prolonged slightly, rough and sharp, whiffing or whipping in quality, is heard,—the more marked, the greater the pressure exercised on the front of the vessel. Such murmur may, however, in rare instances, be heard in the back. Until lately, I supposed that the discovery either of double or of diastolic murmur, in the course of the aorta, positively indicated disease in its coats; but I now know, by a case observed with great interest during life, that there may be intense aortic pulsation, with diastolic murmur, though the vessel is perfectly free from disease, and in calibre below the average. In the instance referred to, however, the pancreas was found, after death, enlarged and hardened, and had pressed somewhat on the vessel in the site of the murmur.†

1470. Theory would lead to the idea, that in cases of pulsation limited to the aorta, the vessel should be the seat of some organic change; else why such limitation? Few examples are recorded of *post-mortem* investigation; in some of the number, affirmation is absolutely made that the vessel was in all respects sound; in others, that its walls were flaccid (but as cause or effect of the pulsation?); in yet others, thin. The calibre of the vessel may be reduced by the pressure of adjacent morbid structure. However produced, the effects of aortic pulsation are sufficiently serious: it increases already existing nervousness, excites apprehensions on the part of the patient of deep-seated organic disease, deprives him sometimes of the power of taking exercise, and interferes with digestion, whence arise loss of appetite and emaciation: and some patients, constantly dwelling on the symptom, magnifying its importance, and fancying that the bystanders must notice it as well as themselves, acquire an utter distaste for all society.

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* In applying the terms systolic and diastolic to mark the rhythm of sounds and murmurs in the arteries, I intend to signify synchronism with the systole and diastole of *the heart*, unless the contrary be stated expressly.

† Eliz. Gosling, U. C. H., Females, vol. v., p. 130.

1471. The duration of aortic pulsation may be very considerable; Baillie thought that once developed, though it might vary in amount, it seldom disappeared altogether: where produced by, or even connected with slight, anæmia, it is positively, however, susceptible of complete cure.*

Aortic pulsation is not so common in ordinary hysteria and spinal irritation as might be expected; it accompanies many utero-ovarian diseases, with pelvic and abdominal neuralgiæ; follows anæmia of all modes of origin; occasionally attends plethora, sthenic and asthenic; and is induced in some susceptible frames, by green tea, strong coffee, tobacco, and similar agents. Various disordered states of the chylopoietic viscera produce it—simple dyspnœa, flatulence, and hepatic disturbances among the number; but special proneness to the complaint probably exists, where causes so slight suffice for its generation. Acute inflammation of the stomach, bowels, and peritonæum, as insisted on by Dr. Stokes, and chronic gastritis, as chiefly illustrated by Dr. Faussett, sometimes produce very marked sympathetic action of the abdominal aorta. Pressure of all kinds directly on the vessel takes an important place among its causes; I have known an accumulation of fæces in the transverse colon induce it in an aggravated form.

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* Brackenbury, U. C. H., Males, vol. v., p. 38, March, 1850. I saw this patient again in the autumn of 1853; he was, practically speaking, free from the complaint.

1472. *Diagnosis.*—A very little care will distinguish pulsation of the aorta in the epigastrium, from the epigastric pulsation of a displaced heart, or an enlarged right ventricle; it is unnecessary to dwell on their distinctive marks. But there may be great difficulty in the diagnosis between functional and aneurismal pulsation of the vessel. In the latter case, however, the pulsation is expansile, heavy, powerful, slow, and appears kept back, as it were, by some restraint behind it, instead of bounding freely forwards; thrill is occasionally to be felt, and a tumor, with dulness under percussion nearly commensurate with its size, is discovered. A harsh, grating, hollow murmur, systolic, of the characters elsewhere described [1506], audible in front, may also sometimes be detected along the spine posteriorly. All this is accompanied with pain more or less tearing or agonising; and the distinction of the cases is not difficult.

But where an aorta, healthy in itself, is pushed forwards by an enlarged vertebra, or a tumor connected with the spine—or where an indurated mass lies in front of, or to either, or both, sides, of the vessel, lateral expansion may be simulated, the murmur may be harsh, though not grating,—while local pain and general emaciation may be produced by the organic morbid state, whatever it is, superadded to the pulsation. Under these circumstances, the difficulty of diagnosis may be extreme. It appears from the case just referred to, that even the discovery of diastolic murmur in the vessel, that is, synchronous with its own systole, will not prove the existence of aneurism positively; still such murmur very rarely attends inorganic pulsation. As a rule, too, the murmur of the latter origin is inaudible in the back; but that of aneurism may be similarly limited in extent: the sex of the patient may give accidental aid; for aneurism of the abdominal aorta is very rare in the female, aortic pulsation common. Again, the existence of inorganic murmur in the heart, thoracic aorta, and veins may assist also; but it may tend to deceive too, for the subject of aneurism is not exempted from becoming anæmic. In ordinary cases, then, the diagnosis is easy; in some rare instances it will be well to watch the case for a time before risking an opinion, and to positively affirm the absence or presence of slight peripheral dilatation of the coats of the vessel (Diag. III., fig. 1, p. 737), may, even after prolonged observation of the case, remain impossible.

1473. *Treatment.*—In the majority of cases, the treatment of aortic pulsation directs itself to the removal of its causes,—such as spanæmia, spinal irritation, gastric or intestinal disturbance, or leucorrhœa.

When the affection is more purely nervous, its management should be conducted as follows:—all suspected articles of food, strong tea, and coffee, and similar stimulants should be forbidden, and a plain, nutritious diet rigidly adopted, with but little vegetable or other substances that promote flatulence. An occasional warm aperient, with some carminative adjunct, is advisable. Anti-spasmodic medicines afford relief to the symptom, though they fail to reach its cause; of these valerian, æther, ammonia, asafœtida, and musk may be employed; sedatives, such as lettuce, conium, and hyoscyamus, may be given in alternation with others of the class acting more especially on the heart, namely aconite, digitalis, and hydrocyanic acid. These medicines are beneficially combined with tonics, where the general indications for such agents present themselves.

The application of some four or five leeches to the epigastrium sometimes distinctly tranquillises the pulsating vessel,—even where no fair suspicion exists of the presence of gastritis in any form. Dry-cupping I have sometimes found beneficial, however difficult of explanation the fact may appear. Anodyne embrocations, belladonna plaster, and the endermic use of morphia in the scrobiculus cordis, moderate the arterial action.

Change of air and travel, moderate exercise, daily friction of the skin, the shower-bath, sea-bathing, or the tepid salt-water bath, and in fact, all hygienic influences, that strengthen the nervous system and improve the health generally, are among the most effectual agents in the cure of obstinate cases.

ACUTE AORTITIS.

1474. Acute aortitis is a rare disease, at least as far as demonstration of its existence goes; it appears to be singularly uncommon, where it might be frequently expected, namely, in connection with acute endocarditis.

1475. *Physical Signs.*—The signs of this affection are obscure,—at least in the present state of knowledge. Violent pulsation of the vessel, and tumultuous action of the heart, sometimes exist; it is very probable that, if lymph be deposited to any extent on the lining membrane, thrill may be perceived, where the vessel nears the surface of the chest. In a remarkable case, observed by Dr. Parkes,* an extremely loud, rough systolic murmur continued audible from the third dorsal vertebra quite down to the lumbar region—a murmur obviously due to the passage of the blood in the vessel over a surface roughened by patches of lymph. In this instance the pulse was irregular and small, but the aortic orifice was contracted, and otherwise diseased, and the heart in a state of dilated hypertrophy; it does not appear that in the uncomplicated inflammation the pulse becomes irregular.

1476. *Symptoms.*—Intense general uneasiness and jactitation are very usual symptoms; general tenderness of the skin has been noticed by Dr. Bright; rigors announce the onset of the disease; and M. Bizot insists much on the significance of general acute œdema of the trunk, arms, legs, and face.

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* Medical Times, Feb. 23, 1850.

Dyspnœa, it appears, may be absent, when the disease is simple; yet it is difficult to conceive that the vessel can be inflamed to any extent without affecting the respiration: generally speaking, other affections, directly implicating the action of the lungs, co-exist. From some observations by Dr. Corrigan, it would appear that inflammation of the mouth of the aorta may induce a series of pseudo-anginal symptoms; but on the other hand, that such is not a necessary effect is shown by Dr. Parkes's case. Pain, with sensation of heat, in the course of the vessel, complained of inferiorly, both anteriorly and osteriorly, on the level of the lumbar spine, has occasionally been a prominent feature. Syncopal tendency and apprehension of immediate death were noticed by Dr. Corrigan. Of the state of the urine nothing is known; yet, as we shall presently see, this is probably a matter of considerable importance.

Dr. Chevers, who has given much attention to the diseases of the vessels, infers from collated cases, that death occurs in cute aortitis with extreme prostration, sharpened or bloated and livid features, cold and discoloured surface, rapid indistinct pulse, stertorous respiration, swollen extremities, and duskiness of the superficial veins; the patients die comatose, and altogether with the aspect of persons destroyed by an animal poison. There is an asthenic variety of the disease, too, in which the ymptoms are adynamic from the outset.

1477. *Diagnosis.*—It will, I think, be generally conceded that the elements of a positive diagnosis of acute aortitis are yet to e found. If narratives may be implicitly trusted to, cases ccur where, supervening on other affections, acute or chronic, cute aortitis produces no obvious effect except increased irritaility and distress: clearly, the diagnosis of the disease could ot, under such circumstances, be ventured on. Pain, thrill, and pulsation in the course of the vessel, with arterial murmur oasting the spine, and answering in localisation neither to a murmur of the aortic nor of the mitral valves, would be the onditions, coupled with great general distress and febrile action, most nearly warranting the diagnosis of the disease. But it is needless to point out the varieties of states that might simulate the entire series, except the aortic murmur; and in respect of this murmur, the possibility of its depending on chronic disease proclaims the necessity of caution.

According to M. Bizot, œdema of the trunk, arms, face, and lower extremities, occurring acutely, without functional disturbance of any organs but those of the circulation, indicates acute aortitis. But, on the one hand, I have seen the anatomical evidences of acute aortitis, where no anasarca had occurred;* and, in like manner, Dr. Parkes's case, one of the most indubitable on record in regard of anatomical characters, proves absolutely that the disease may exist in perfection without any such œdema: while, on the other hand, M. Bizot has not taken into consideration the state of the kidneys in his patients. Now, in one of the three, Bright's disease appears to have been positively present; and in the other two we have no assurance that it was absent. M. Bizot's observations were made before renal diseases were either clinically or anatomically paid much attention to.

1478. *Prognosis.*—Aortitis may prove very rapidly fatal; it has certainly destroyed life in three or four days in association with other less serious states. The transparence of exuded lymph furnishes the best measure of the recency of the disease. Of the prognosis, all that can be said is, that as persons die of various diseases with the evidences of chronic inflammation in the vessel, either acute aortitis sometimes fails to kill, or chronic aortitis sometimes pursues a chronic course from the outset: both propositions are probably occasionally true.

1479. *Treatment.*—Were the disease upon fair grounds even suspected, active measures should without delay be had recourse to. Venesection or free cupping in the course of the vessel in the front of the chest and along the spine, or if the disease have occurred in a very low state of system, dry-cupping, are clearly indicated. Counter-irritation, by the application of a long narrow blister along the left vertebral groove, which may be used also for the purpose of applying calomel and morphia endermically, has theoretical argument in its favour; for the mass of tissues, intervening between the skin and vessel, is in all probability sufficiently great to render the effect of the blister antagonistic.

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* Campion, U. C. H., Females, vol. vi., p. 41.

The internal medicines, deserving of most confidence, are calomel, opium, and tartarised antimony : if the excitement of the heart and vessel were very great, digitalis or acetate of lead might be simultaneously administered. Aperients and saline diaphoretics are worthy of attention as adjuvants.

In employing these and other measures the practitioner must never lose sight of the constitutional state.

CHRONIC AORTITIS.

1480. Thickening of the coats of the aorta, undue vascularity of the outer membrane, unevenness, roughness, puckering, furrowing, and channelling of the inner surface of the vessel, with various alterations in its calibre, producing irregular distension and contraction, constitute unquestioned characters of chronic inflammation. White opaque cartilaginiform patches, studding the inner surface of the vessel, are likewise admitted to be the chronic representatives of the lymph-exudations of the acute disease. The relationship of saline precipitation to these patches is made matter of dispute ; the fact appearing to be, that, while the usual nidus of calcification is certainly atheroma, the white patch is the occasional seat of the change, either primarily, or secondarily to the deposit of atheroma within itself.

1481. But, serious as these conditions are anatomically, we know of no symptoms to which they give rise, unless they have led to, or, at least, are attended by, very considerable alterations of calibre of the aorta, in the forms of obstruction, coarctation, or dilatation,—conditions which will presently be more fully considered. Nor is it readily conceivable, indeed, *à priori*, that much local disturbance should be produced by these changes. Pain of a notable kind can scarcely be expected; and until the elasticity of the vessel has been very deeply impaired, it continues capable of taking its part—in great measure a mechanical one—in carrying on the circulation.

ATHEROMA AND CALCIFICATION.

1482. Atheroma and calcification of the aorta, conditions of great anatomical interest, have in themselves but little clinical importance. Destroying the elasticity of the vessel, rendering it fragile, contributing to the production of various aneurismal changes, and connected with two important diatheses—the fatty and the calcifying—the prominence of these conditions of the vessel can scarcely be overrated in a pathological point of view: and in their ultimate possible influence they are of grave clinical interest; for calcareous matter may be the occasion of obliteration of the vessel, by protruding more or less into the interior, and affording points for the blood to coagulate around. Yet, until such obstruction more or less prominently displays itself, what symptomatic indication have we of atheromatous or fatty deposition in the coats of the aorta? None, certainly, in the present state of knowledge. And of physical evidences there are none, except systolic rough murmur in the course of the vessel, either limited thereto, which is rare, or audible also in a less intense form at the aortic valves [503],—jerking, inelastic impulse behind the sternal notch, and, occasionally, systolic thrill both at that point and about the second right cartilage.

COARCTATION AND OBLITERATION.

1483. I. The arch of the aorta is subject to a peculiar *quasi-congenital* coarctation which, occurring at the time of the closure of the ductus arteriosus,* is connected immediately in position with that duct, may pass on to total obliteration, produces in the great majority of cases but little clinical ill effect, and has never, so far as I know, been positively diagnosticated during life.

1484. In some cases symptoms resembling those of aneurism of the arch have been noted, dyspnœa, palpitation, and pain behind the sternum: œdema of the ankles does not appear among those mentioned, unless where enlargement of the heart or valvular disease co-existed. Violent throbbing of the carotid and temporal arteries, an occasional symptom, is explicable by the enlarged calibre of those vessels.*

1485. Loud systolic murmur, of maximum force at the second right cartilage, but greatly more intense at the upper part of the sternum generally than at the cardiac region, where it is evidently audible only by transmission, accompanied with well-marked thrill in the second interspace on each side of the sternum, and also above this bone, may be expected, on the evidence of two cases reported by Dr. Blakiston, to attend this coarctation of the vessel. The amount of thrill will probably vary with the precise position of the arch, the condition of its inner surface, and the state of the blood. Highly-marked aortic systolic murmur and thrill will not then justify positively the inference of aneurism: some of the other physical signs of the latter disease, such as local prominence and dulness under percussion, or some of the class of concentric pressure-signs, are required in addition to warrant its diagnosis. Anæmic blood, rushing past the inequalities of surface, caused by irregular calcification of the arch, might give rise to a murmur limited in situation to that portion of the vessel.

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* This closure, commencing after a few respirations, is, according to Bernt, complete, in some cases, by the third or fourth day, by the eighth day in half the number, and by the tenth in all.

* Nothing can be more curious than the anastomotic enlargements that occur in these cases. Carswell's Drawings, U. C. Museum, A. 20, exemplify this: here the innominate artery is considerably thicker than the arch, the arteries of the base of the neck greatly enlarged, and nameless ramusculi as full as the radial artery in the natural state; the internal mammary and epigastric arteries are especially of large calibre.

1486. If, then, an aortic murmur, barely audible at the heart, were discovered as a permanent condition in a person free from spanæmia, and if there were neither undue dulness under percussion nor bulging of the surface in its site, and the patient suffered neither from the local or general symptoms, nor the concentric pressure-signs, of aneurism or tumor, there would be fair motive for strongly suspecting the existence of coarctation of the arch. This view would be greatly strengthened if the right carotid, sub-clavian and subsidiary arteries, were obviously enlarged and prone to violent throbbing; otherwise all the conditions enumerated might depend on an obstruction at the aortic orifice of the innominate artery.

1487. Of seventeen recorded cases fourteen are furnished by males, three by females. Of sixteen persons, dying with such coarctation, two were under ten years of age, eight aged between ten and forty, five between forty and sixty, one between sixty and seventy.

1488. The possibility of living on in excellent health with a coarctation of this kind is amply proved by several of the cases just referred to. Adult, nay aged, patients, have been cut off by various diseases, in whom it had existed to a very high degree without having ever attracted attention during life to the organs of circulation: its existence has, in truth, been wholly a *post-mortem* discovery. On the other hand, violent efforts, by over-filling and straining the thoracic organs of circulation, may at any time cause sudden death. Under such circumstances, rupture of the aorta on either side of the ductus arteriosus, and rupture of the left ventricle and right auricle have been observed.*

1489. II. The calibre of the aorta may undergo diminution of an *acquired* kind, as a result both of its own diseases, of certain affections of the heart, of the lungs, and of others of the system at large. Under the first head appear contractions depending on exudation-matter connected with the coats of the vessel. Under the second, range themselves reductions of size depending on the closely constrictive action of induration-matter in the pericardium, and on obstruction of the mitral orifice, which entails a deficient supply of blood to the aorta. Next, prolonged obstruction of the pulmonary circulation acts in a somewhat similar way: the calibre of the aorta is below the average in persons cut off with vesicular emphysema of long duration, unless the right ventricle have become the seat of dilated hypertrophy. Lastly, in cancer and phthisis the vessel suffers in the same manner, probably from the gradual reduction in the amount of circulating fluid in those diseases.

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* Crisp, Diseases of the Blood Vessels, p. 31.

1490. III. A considerable number of cases are on record in which imperviousness, more or less absolute, of the aorta has been produced in the adult by coagula forming around prominent spiculæ of calcification, or by the products of inflammation, aided by contraction of the walls of the vessel itself. The lower part of the thoracic and the abdominal aorta are the most common seats of the disease.

1491. The course of this affection, symptomatically, may be acute or chronic. In the former class of cases, doubtless, the current of blood, though it may long have been somewhat obstructed, has not become seriously interfered with until the appearance of acute symptoms,—these symptoms being dyspnœa, anasarca, tendency to gangrene of the lower extremities, and hæmoptysis. But as the state of the lungs and heart is imperfectly known in the very few instances of the kind, the direct dependence of hæmoptysis on the obstruction may be questioned.

1492. In 1835, I saw in the wards of M. Louis a remarkable example of obliteration of the abdominal aorta, in a female aged fifty-one. Here, four years previous to death, numbness, first of the right, and, some months later, of the left, lower extremity, were clearly the earliest positive effects of the growing obstruction. These were followed by inability to walk, not from fatigue or cardiac suffering, but from pain, coldness, and increasing numbness in the legs. Subsequently, the effects of organic disease of the heart (she had mitral constriction, dilated hypertrophy of the right ventricle, palpitation, pulmonary apoplexy, and hæmoptysis) threw those of the aortic obstruction into the shade, and eventually cut her off in a few days after admission to the hospital. Twice in the course of her illness she had slight and passing œdema of the lower extremities, but there was none on her admission, and at death scarcely any. Besides the local obliteration, the calibre of the aorta generally, and of its branches, was below the average in this woman. No attempt appeared to have been made at anastomotic enlargements; and the disease had, to all seeming, originated in inflammation of the vessels.

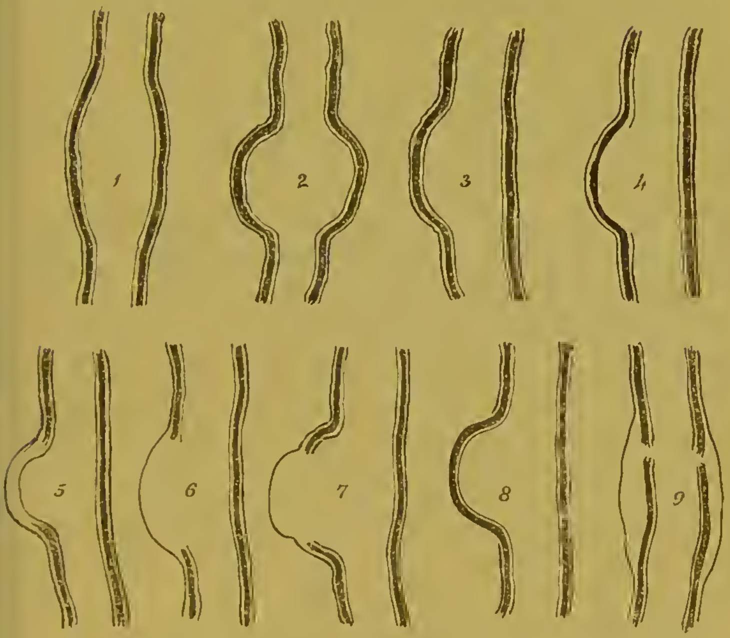
ANEURISM OF THE AORTA.

1493. I. The term "aneurism," understood in its widest sense, may be defined as a local increase of calibre of an artery. And in this sense it has been used by some authors, while others have made attempts to restrict its application in many different ways. Professional opinion is, indeed, so unsettled as to the proper application of the term, that an explanation of the sense he, in particular, may attach to it, is called for on the part of every person employing it.

1494. Adhering, then, to the comprehensive definition above expressed, I would divide the *genus* Aneurism into the subjoined *species* and *varieties*:—

A. Peripheric: *dilating*	Fusiform. Globular.
B. Lateral: *sacculating*	a. Simple. b. Compound. c. Mixed.
C. Interstitial: *dissecting.*	

1495. The anatomical constitution of these varieties of ieurism is exhibited to the eye in Diagram III.



g. 1, Peripheric Dilating Aneurism; the three coats of the vessel all round *gradually* widening, so as to give a *fusiform* shape to the enlargement. Fig. 2, Peripheric Dilating Aneurism; the three coats of the vessel all round *abruptly* widening, so as to give a *globular* shape to the enlargement. Nos. 1 and 2 are the "simple dilatations" of various authors. Fig. 3, *Simple Lateral* Sacculating Aneurism; the Sac, formed of the three coats of the vessel unbroken, rises from a limited portion of its circumference: the "*true* aneurism" of some authors. Figs. 4, 5, 6, 8, varieties of *Compound* Sacculating Aneurism; all agreeing in the characters of lateral sacculation, and injury to the coats of the vessel: the "false aneurism" of writers. Fig. 4, the Sac formed of the middle and outer coats, the inner being destroyed. Fig. 5, the Sac composed of the outer coat lined by the inner, the middle tunic having disappeared. Fig. 6, the Sac, composed of the outer membrane alone. Fig. 8, the Sac, composed of the middle and inner coats, forming a sort of hernial protrusion through the destroyed outer coat. Fig. 7, Mixed Aneurism, a combination of the simple and compound sacculating varieties; the simple condition of Fig. 3 having existed for a variable time, the middle and inner coats more or less suddenly give way, and the condition exhibited in Fig. 6, is added to the original simple disease. Fig. 9, Dissecting Aneurism; separation of the outer coat from the middle, by blood escaping from the interior of the vessel, through fissures in the lining and middle tunics.
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1496. Now, in the aorta, the only vessel with which we have here to do, certain of the above varieties are distinguishable from each other by symptomatic characters, and, in reference to this important practical fact, may be placed in four *clinical* groups, thus :—

(*a*) The simple and compound lateral sacculating aneurism of figs. 3, 4, and 6 ; (*b*) the fusiform and the globular dilating aneurisms of figs. 1 and 2;* (*c*) the mixed aneurism of fig. 7 ; (*d*) the dissecting aneurism of fig. 9.†

By far the most important of these groups is the first. Its constituent varieties in the present state of knowledge are not distinguishable by any positive signs during life ; hence their association in this clinical scheme.

1497. The effects, signs, and characters of aneurisms of the aorta differ so much in the chief divisions of the vessel, that the separate consideration of the disease in the arch, with the ascending aorta conjoined, in the descending thoracic and in the abdominal divisions, is matter of absolute necessity.

I.—ANEURISM OF THE ARCH OF THE AORTA.

1498. GROUP *a.—Simple and Compound Lateral Sacculating Aneurisms.*—Affect a portion only of the circumference of the vessel; are generally narrower (the compound much more so than the simple) at the neck than at the body of the sac; rarely spring from the inferior curvature of the arch ; range in size from that of a nut to that of the fœtal head; and are filled to variable extents with laminated coagula,—the compound more extensively, as a rule, than the simple, because the orifice of its sac is comparatively narrow. The influences of these aneurisms must seriously differ, according to the precise direction in which the sac chances to enlarge; but a grand practical distinction of specimens belonging to the present group, may be established in this point of view, into the centrifugal and the centripetal,—those tending to grow outwards towards the walls of the chest, or inwards towards deeply-seated parts. The former habitually attain much greater bulk than the latter, for the simple reason that organs of vital importance being secure from pressure, the essential functions are, comparatively speaking, but little interfered with, life is prolonged, and time given for the free enlargement of the sac.

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* Globular dilating aneurism, apparently most common in the cerebral arteries, I had never seen in the aorta until quite recently,—May, 1852. The specimen is now placed in the U. C. Museum, No. 4036.

† I strongly doubt that the sac, in fig. 5, is such as it has been represented to be by authors,—namely, the outer coat lined by the stretched inner membrane. The apparent inner tunic is probably nothing more than a film of adventitious formation ; at all events, I have never seen a sac so constituted. Of fig. 8, there is but one positive example known, so far as I can ascertain,—namely, the preparation, No. 1642, in the Hunterian Museum. Another variety of hernial aneurism, protrusion of the inner through the middle and outer coats, has been described : in all probability, imaginatively.

1499. The effects of an aneurismal sac on the adjacent parts are of various kinds; but all, directly or indirectly, due to pressure. Mere *displacement* is illustrated by detrusion of the trachea, or œsophagus, sidewards or backwards, of the heart downwards; by anterior bulging of the walls of the chest; by dislocation of the sternal end of the clavicle; and by depression of the apex of the lung.* *Interference with the freedom of hollow canals* is seen in obstruction of the œsophagus, the trachea, either large bronchus, the descending cava, the innominate, left carotid, or left subclavian arteries,—vessels which, blocked up either by direct pressure, by twisting of their orifices, or by coagula, become more or less functionally disabled. *Injury to parenchymata* is exhibited in compression of lung-substance, condensed so as to be incapable of expansion. *Destructive and perforative absorption of tissue* may occur in the trachea, the œsophagus, the pleura, lung, and bronchi; the pericardium; the pulmonary artery; the substance of the lung itself; the thoracic duct; the ribs,* sternum, rarely the clavicle, the vertebræ, though aneurism of the arch seldom, however, actually makes its way into the spinal canal; the recurrent nerve, and the spinal nerves on the left side. *Exudation of lymph* often takes place between the aneurismal sac and the parts pressed on, as the ribs and sternum, the pleura and lung. *Irritation of nerves* is exhibited in various spasmodic phenomena connected with traction of the recurrent nerve. And, lastly, actual *inflammation of texture*, as an indirect result of pressure, is displayed in the tracheitis and bronchitis that so often accompany the disease. Now, as will be readily understood, many of these different effects are clinically useful in guiding the observer to a precise localisation of the disease. Further illustrations will be found in the description of mediastinal tumors [1104—1109].

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* Brader, U. C. H. Females, Clin. Lect. "Lancet," loc. cit., p. 117. No lung-substance reached higher, in this instance, than the second rib on the affected side. The aneurism sprang from the descending portion of the arch, just at its union with the transverse, and was combined with small fibro-fatty tumor in both mediastina.

1500. *Physical signs.*—The physical signs of a sacculated aneurism of the arch are in well-marked cases extremely numerous, and as precisely and directly significant as, perhaps even more so than, in any other disease. But, on the other hand, the instances are rare, where the signs are numerously associated, unless the dilatation have attained very considerable dimensions; any one of the series of signs may be absent throughout the course of the disease; and sometimes during its earlier periods there is not a single one of the number satisfactorily developed.

1501. (*a*) By inspection, local bulging is discovered, tending, when at all notably prominent, to the gently conical form. The ribs and interspaces equally contribute to its formation, when the area of the bulged surface is at all extensive; but when of limited extent, that is, at the time the sac commences first to

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* Small aneurisms destroy contiguous bone more readily than large ones, obviously because pressure is more concentrated in the case of the former.

ict on the surface, a single rib or cartilage may alone be rominent, or the end of the clavicle simply pushed forwards. In cases of long standing, and where the base of the conical ulging is extensive, the skin becomes smooth and glazed-ooking, the unevenness of the ribs and interspaces being ompletely removed. Concerning the seat of this bulging: f the sac spring from the first division of the arch including vhat is called the ascending aorta, the prominence mainly ppears at the right edge of the sternum, in the second nterspace and second cartilage, up to the first, and even as ar sometimes as the clavicular joint. But if, while the sac nvolves this division of the arch, it likewise implicates the ransverse and somewhat the descending portions, the prominence, instead of lying to the right, may be placed solely to the eft of the sternum, in the infra-clavicular and mammary regions, xtending as far outwards as the line of the nipple;* obviously he ascending part of the arch gets twisted to the left. Secondly, f the transverse division be the affected part, the top of the ternum, or the right first cartilage form the prominence; but f the sac be of small size, actually limited to this segment of he vessel, and springing from its posterior aspect, there may be, r, more correctly, will be, no visible bulging at all. Thirdly, hen the descending portion of the arch is sacculated, the rominence appears at and about the second left cartilage: but neurisms thus situated do not often cause anterior bulging; he vessel is here far away from the front of the chest, and, as atter of experience, the tendency of its aneurisms is to enlarge o the left side, while they expend their force posteriorly in roding the vertebræ. Whatever be its seat, the superficial xtent of the prominence is less than the diameter of the sac; e bulged surface corresponds to the most prominent part of

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[*] Harris, U. C. H., Males, vol. ii., p. 262. The sac in this remarkable se commenced two inches above the aortic valves, and terminated close to e left subclavian; the innominate artery was carried to the left, so as to lie front of the origin of the left carotid.

the latter. The actual area varies between the size of half-a-crown and that of a large cocoa-nut. Deficiency of bulging is rarest where the sac lies to the right; commonest, where it lies to the left; of medium rarity, where the horizontal and central portion of the arch is implicated.

Movement, pulsatile, expansile,[*] and synchronous with the heart's systole, may be seen in the bulged part of the anterior chest; and, in some cases, motion of more abrupt and non-expansile character is very perceptible to the eye above the clavicles, evidently coming from below and as evidently produced by a body of some size. But if a sac, even though of massy bulk, be filled in great measure with fibrine, while a dwindled current of blood trickles with more or less difficulty through a small channel at the furthest aspect from the anterior surface of the chest, that sac may, especially if seated in the descending part of the arch, be as absolutely pulseless to the eye as if totally unconnected with the arterial system.

1502. (*b*) The hand, applied to the bulged surface, appreciates more accurately the motion of the sac,—generally a very little behind the apex-beat of the heart in point of time, its systolic impulse is sometimes, as far as the sense of touch can determine, synchronous with this. By systolic impulse we mean that synchronous with the systole of the heart: if the vessel alone were considered, this impulse would of course be called diastolic. In certain instances, by no means in all, the impulse is double, a receding as well as an expanding motion existing.[†] The force of the systolic impulse is sometimes extreme; even from a sac of small size, and mainly seated behind the sternum, which may have undergone but very slight erosion, the throb may be sufficiently strong to shake the head applied to the stethoscope.* In character, throbbing simply or thrusting and heaving, dull and inelastic (the latter condition marked in proportion to the quantity of fibrine in the sac), there are cases in which it is very positively undulatory. When the walls of the sac reach directly to the surface,—when they have bulged this considerably,—when there is a free passage through the sac,—and when laminated coagula in but small quantity lie in its interior, the current of the blood passes with wave-like motion directly beneath the finger laid on the skin.† The seat of palpable is the same as that of visible action. When the descending portion of the arch is aneurismal, the impulse of its transverse division, which we will suppose sound, may be considerably intensified, as felt by a finger placed in the sternal notch; or it may vary from day to day, independently of any concomitant change in the impulse of the prominence directly over the sac, —a circumstance sufficiently calculated to puzzle the observer as to the real seat of the disease. Aneurisms seated in this part of the arch tend also to raise its transverse division slightly above the natural level. In rare instances, very positive impulse may be detected in the inter-scapular region, more frequently on the left side than the right,—a fact explained by the relationship of the arch on the two sides to the spinal column. Sometimes unnatural impulse in the region of the arch may be detected by laying one hand on the top of the sternum, the other in the intra-scapular region,—the single hand having failed to distinguish anything abnormal.

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[*] The expansile character of the pulsation was shown to the eye remarkably in the case of Warren, U. C. H., vol. viii., p. 25, 1849. A belladonna plaster had been placed over the aneurismal bulging, which was remarkably broad-based; as the plaster produced an uncomfortable feeling of tension at first, it was slit across horizontally; subsequently, with each throb of the sac, the edges of the incision diverged by about a line.

[†] It is perhaps incorrect to speak of this aneurismal motion synchronous with the systole of the vessel, as an impulse,—it is rather a receding jog.

Systolic vibratile thrill of variable amount may attend the impulse, or be perceptible even where distinct impulse cannot be detected. In aneurisms of the group under consideration, thrill is less constant than in the group of peripheric dilatations; but I have known it intensely marked, sufficiently almost to tickle the hand. Thrill may be limited to the bulged surface, extend slightly in all directions beyond its confines, or be perceptible above the clavicles and sternal notch. The sign may be more distinct in the latter situation than over the sac itself. Thrill may exist at one time, disappear and return more forcibly than ever,—a fact in some instances explicable by the varying state of the blood in respect of anæmia, in others referrible to changes in the state of the sac, its contents, and its inlet.*

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* Downie, U. C. H., Males, vol. iv., p. 273.

† Harris, U. C. H., Males, loc. cit.

The vocal fremitus may be annulled, over a sac of medium size, under the right clavicle.†

1503. (*c*) Aneurismal sacs, according to the direction in which they point, increase the measured distance from the sternal notch to the nipple, the middle line to the nipple, or both. They interfere with the measurable chest-expansion of breathing on the surface corresponding to themselves, and may restrain this very greatly by pressure on a chief bronchus.

1504. (*d*) Dulness, with resistance, under percussion, exists in the surface nearest an aneurismal sac. The reasons why the superficial extent of dulness should prove, practically speaking, less than that of the aneurismal pouch and artery, from which it springs, combined, have already been explained [490]. An area of some square inches may be rendered dull by sacs of very large dimensions. The observer must always bear in mind the possibility also of the real dulness of a sac being factitiously increased in extent by adjacent consolidated lung, or solid accumulation in the mediastinum.

The seat of dulness in *front* of the chest bears the same relationship, as that of prominence, to the different portions of the arch. As a rule, it is most easily and completely detected to the right of the sternum and in connection with the ascending portion of the arch and its angle on the right side; but if twisting of the vessel take place, the deficiency of resonance, even though the ascending portion of the arch be implicated, strikes us most obviously to the left of that bone.* Nor can it be said that a sac springing from the descending portion of the arch, even when small, fails to effect the percussion-sound; but, for obvious topographical reasons, it does so proportionately less, both in superficial extent and in amount. A sac originating in the horizontal part of the arch impairs the natural osteal clearness of the resonance immediately below the sternal notch, the edges of the lungs being pushed sidewards to a variable extent: it also affects the sound above and to either side of the bone. Whatever be the seat of the pouch, it is important to ascertain positively whether the dulness connected with it does, or does not, reach completely into the acromial angle of the infra-clavicular region. Aneurisms of moderate size seated in the horizontal and descending parts of the arch give dull sound *posteriorly* in the inter-scapular regions; and a large sac, even though derived from its ascending portion, will impair the resonance between the right scapula and the spine.

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* *E. g.* Case of Harris, loc. cit.—Thrill disappeared December 4. returned December 21.

† Mr. ——, seen with Mr. Tapson, October, 1852.

If a sac be moderately stratified internally with fibrine, the character of its resistance is nowise special. If, on the contrary, it be closely filled with such coagula, and the amount of fluid blood within it be small, the resistance is dull, inelastic, and putty-like. This kind of resistance is significant enough, when discovered; but it is scarcely necessary to add, that either for the purpose of eliciting this, or any other, character of percussion, the least roughness in manipulation is not only awkward and *unclinical*, but actually dangerous.

In a former place [490], I have touched on the question of the smallest amount of dilatation of the arch capable of being demonstrated by percussion. Under a concourse of favouring circumstances, we have seen that a very small increase of size may be so discovered. The inefficiency of percussion increases with the smallness of the sac and its distance to the left of the median-line. There can be little doubt that more than one small aneurismal sac, in the middle division of the arch, has escaped detection, simply because percussion was not performed below, and at, the sternal notch.

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* Harris, U. C. H., Males, loc. cit.

1505. (*e*) Few diseased states give rise to such variable auscultatory signs, as a sacculated aneurism of the arch,—a fact sufficiently proved by the following series of conditions of sound that have actually fallen under my notice: the list probably might be increased from the experience of others. Through the stethoscope, placed on the most prominent part of the surface, I have heard—

1. A double sound, both divisions of which are rendered murmur-like by suspension of the respiration, and both are weaker than the sounds at the base of the heart. 2. A double sound of the same characters, except that its divisions are louder than the basic sounds of the heart. 3. No sound at all, properly speaking; but a dull, impulsive impression, systolic in time, that simulates sound. 4. A systolic impulsive impression of the kind, with a clear ringing second sound.* 5. A systolic blowing murmur, harsh in quality—harsh to the same, a greater or a less amount than elsewhere in the artery close to the sac; with a dull muffled diastolic sound. 6. A roaring, grating systolic murmur, stronger than at the base of the heart, the aortic valves being constrictively diseased, and the blood spanæmic; with a diastolic sound. 7. A double rough murmur, the systolic division louder than the diastolic. 8. A double rough murmur, the diastolic division louder than the systolic. 9. A systolic sound, with a diastolic murmur,—a very rare combination.†

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* Bell, U. C. H., Males, vol. vii., p. 168.

† It is to be understood of all these varieties of aneurismal murmur, that

Aneurismal sacs may then be, as it were, silent; or be the seats of sound or murmur, either singly or both combined. On the relative frequency of these different acoustic states I can make no positive assertion; nor do the elements for such assertion exist anywhere, that I am aware of. But of one fact, that murmur is vastly less frequent than the language of the majority of writers would indicate, no one, accustomed to clinical observation, entertains the least doubt. Another fact not generally known, or at least habitually lost sight of, is that the conditions of sound or murmur over a sac not only vary at different periods of the disease, but even occasionally, from day to day, in different postures, and at different phases of the respiratory act.

1506. The sounds heard over aneurismal sacs are generally speaking identical in special character with those of healthy arteries,—they are simply more intense. But occasionally, a peculiar character in the single or double sound of an aneurismal sac, when completely free from murmurish quality, may be perceived, that seems best designated by the phrases *pumping* or *sucking:* this variety is not without diagnostic significance.

Of the characters of aneurismal murmurs, the following are worthy of clinical attention. The quality of the *systolic* murmur may be simply blowing, or blowing with a peculiar hoarse hollowness, which, when well marked, is important in diagnosis, grating, rasping, sawing, filing, or if the blood be spanæmic, roaring. Variable, but generally low, in pitch, this murmur may be of higher pitch, nevertheless, than a co-existent systolic murmur at the base of the heart. Short and abrupt in the majority, prolonged, almost drawling, in the minority of cases, ts intensity may reach, or even exceed, that of the loudest murmurs produced within the heart. The strength of the *diastolic* murmur, though variable, is rarely great, absolutely speaking: in quality, it is generally softer than the systolic;

he influence of valvular disease in their production is, as matter of observaion, supposed to be excluded.

it is not constantly present, not only at different periods of the same case, that has at one time presented it, but even with successive beats of the heart. Generally best audible over the most projecting point of the prominence, murmurs may, on the contrary, attain their maximum loudness at the edges of this: or, even when the main part of the aneurism lies considerably below the clavicles, they may be strongest at the base of the neck,—probably, under the latter circumstances, thick layers of fibrine are accumulated in the lower parts of the sac: or, lastly, they may, in very rare cases, exhibit greatest intensity on the left side of the spinal column.

The mechanism of these murmurs seems to be this. The systolic is either produced by the passage of blood over a surface roughened by fibrinous masses, inequalities in the coats of the vessel, and calcifications; or it may come of the rippling motion given to the fluid on its entry from a portion of tube of natural calibre into one more or less dilated, especially if the dilatation be abrupt; or it may be caused by the flow of blood through the comparatively narrow and more or less rough orifice of the sac; or not directly dependent on the aneurism itself, it may proceed from a spot of the vessel pressed upon and rendered narrow by the sac. On the other hand, diastolic murmur seems due to the reflux of blood from the sac through its orifice; and as the force of the reflux current must be comparatively slight, so, as a rule, diastolic aneurismal murmurs are feeble: sometimes, where undue force is given to the back current by co-existent aortic regurgitation, the diastolic murmur, especially if the sac be very close to the origin of the aorta, acquires unusual intensity. When the entry of the blood into the sac is murmurless, and its escape productive of murmur,—that is, when diastolic murmur alone can be heard;—the peculiarity probably depends on some special condition of the orifice of the sac, whereby a smooth surface is presented to the entering blood, and a surface, roughened by moveable fibrine or otherwise, opposed to the receding current.

A number of conditions tend either to enfeeble or intensify aneurismal murmurs. If the heart's action be very weak; if the sac be filled in great measure with fibrine; if it be incapable of much expansion; if the opening be very large, inasmuch as the current is then too free; or if the opening be at once very narrow and smooth, inasmuch as possibly the current is then too small to generate notable sound,—if any one of these conditions be present, a sac, otherwise well constituted for the purpose, may fail to furnish murmur. On the other hand, great roughness of surface, neighbouring pressure, and the presence of good conducting material round the sac, will intensify, really or apparently, these murmurs. Mere sounds may sometimes be rendered murmurish by suspension of the breath for a moment,—a fact to which it is not easy to supply a satisfactory clue.

1507. A murmur may be suddenly generated in a person, known subsequently to be the subject of aneurism of the arch, be audible not only to himself, but to bystanders at some distance off, pervade the entire arterial system, though of maximum loudness at the upper part of the chest, and disappear, at least as a phenomenon audible without the aid of auscultation, mediate or immediate, as suddenly as it came.* More than one hypothesis readily suggests itself in explanation of this singular condition; but, as I have no *post mortem* evidence, proving the reality of any one possible mechanism rather than of another, I abstain from conjecture.

1508. *Symptoms.*—(*a*) The weight and flesh of aneurismal patients undergo very considerable reduction in a certain share of cases; while instances, where the sufferer continues stout to the last, or almost to the last, are far from very uncommon. The centripetal or centrifugal process of the aneurism does not always explain this difference; if, on the one hand, the greater disturbance of important functions, attached to the former mode of progress, tends to produce rapid emaciation, the protracted, though less acute, functional mischiefs endured in the latter, eventually work out the same result. Besides, these two topographical modes of progress are sometimes so mixed up, that in not a few cases it is impossible to place the disease exclusively in one or the other category. But from the analysis of seventeen cases of aneurism of the arch, of which I have notes, the inference clearly flows, that the presence or absence of pain of serious character is the real element in determining, or warding off, early emaciation. And that pain is very decidedly greater in cases where the dilated vessel bears either against the chest-wall and intercostal nerves, or the spine and vertebral groove,—hence where the course is centrifugal. It appears that extreme emaciation has sometimes been mechanically caused by pressure on the thoracic duct. The face, trunk, and limbs waste *pari passu* [994].

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* Facts all illustrated by the case of Mr. ——, seen with Mr. Pollard of Brompton: death occurred by rupture of the sac.

There is not any attitude, posture, or mode of decumbency peculiar to the subjects of aneurism of the arch, as a class and for a permanency; in bed the patient usually lies on the back, with the head moderately high. But for the relief of particular kinds of pressure, peculiar attitudes may be assumed; thus, where a sac presses on the trachea, the patient steadily keeps the head forwards, or forwards and sidewards, — and also frequently raises or throws back the head suddenly, keeping it in this posture for a time, so as to project the sac forwards from the windpipe.* When paroxysmal attacks of dyspnœa, from tracheal pressure or irritation of the recurrent nerve, occur, the sufferer sits up with his head supported on his hands, the elbows resting on the knees, or bends over the back of a chair, &c. The sleep is not affected by the aneurism itself; but if pressure exist, the ordinary slumbers are fitful, interrupted by starts, and frightful dreams. During the urgency of bronchitic and asthmatic seizures, the patient may pass night after night out of bed. The easiest attitude in sleep may be one which to a healthy person would prove unbearable: thus with the head supported sidewise wholly on the hand;* postures of the kind may also be assumed consensually during sleep [1110].

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* In a case recently seen, the existence of this habit, combined with slight gnawing interscapular pain, drew my attention to the arch of the aorta, where, with much difficulty, the physical signs of a small sac were elicited.

The expression of the face varies: it may be calm and not indicative of suffering, except during paroxysms of dyspnœa, growing then, terrified and imploring; or habitually cross and irritable; or anxious and worn; or simply significant of profound distress. The differences in the original temper of patients modify their facial expression during this, as all other chronic diseases. The colour of the face may be to the last florid in the main, with slight lividity;† or habitually livid; or in no single point remarkable; or pale, sallow, and cachectic-looking: the latter alone is in the least degree distinctive.

1509. (*b*) The skin of the general surface, though sometimes sallow, cannot be said to exhibit any habitual special tint. Sweating, the reverse of an ordinary symptom, I have nevertheless known give considerable annoyance, where the lungs were sound. The lower extremities remain singularly free from œdema; a fact often finding its diagnostic application. I have known slight bulbousness of the finger-ends, confined to the side on which the supply of blood was limited through the influence of the sac.‡ Œdema of the base of the neck, the face, and the upper extremities, and one or both sides of the thorax, follows pressure on the superior cava, or one or both innominate veins. The peculiar spongy elastic fulness of the base of the neck, looking like a *collar* of flesh, due to capillary turgescence, is also observed. The integuments over the sac sometimes alone become œdematous, from irritation or mere distending pressure. The temperature of the arm, of which the circulation is impeded, may fall very notably below that of its fellow.‡

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* King, U. C. H., Males, vol. vii., p. 89.

† Brader, U. C. H., Females, loc. cit.

‡ Mack, U. C. H., Males, vol. ix., p. 210.

1510. (*c*) The joints are not affected; no positive connexion exists between either rheumatism or gout and aneurism.

1511. (*d*) The lips full, tumid, and livid, the tongue œdematous at the edges, and of purplish tint, the mucous membrane of the pharynx, thick, livid, and coated with viscid secretion, when other signs of venous pressure exist, display, if these be absent, no peculiarity of appearance. Dysphagia, slight or severe, felt more in some postures than in others, paroxysmal or permanent or both combined, or in some severe cases disappearing completely for a while, after having been a constant condition (a change sometimes explicable by diminution of pressure through hæmorrhage from the sac), is a symptom of considerable frequency. More commonly attending the disease in the descending and transverse parts of the arch, dysphagia may be absent even in the former case,* and co-exist with a medium-sized sac springing from the right angle of the arch.† The intensity of dysphagia obviously depends in the main on the general and local nervous susceptibility of the patient; a slight amount of pressure on the œsophagus will produce greater difficulty of deglutition in some persons, than actual perforative destruction of the coats of the tube in another.‡ Blood may be discharged in large quantities, from rupture of the sac, into the œsophagus,—an event which, moreover, need not prove immediately fatal. Whether slight oozing of blood may take place by filtration into the œsophagus, and be discharged by sputation, without actual hæmorrhage, I do not know from experience; but there can be no reasonable doubt of the possibility of the occurrence. The appetite fails altogether, if the neurism be the source of pain; commonly it is capricious. Blood in small quantities, darkened and otherwise altered by he gastric fluids, is occasionally vomited, after having trickled nto the stomach from filtration through the walls of the sac nd œsophagus; blood of similar origin may also be traced in he stools.* The bowels are habitually constipated in advanced ases, probably from the patient's inability to take exercise; latulence distresses many, even male, patients; piles and ruritus about the anus seem to be more frequent than in the verage of persons of equal age. Ascites does not occur.

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* Brader, U. C. H., Females, loc. cit.

† Downie, U. C. H., Males, loc. cit.

‡ A perforation the size of a shilling may exist without the very least dysphagia occurring during life; though, too, the current of blood in the sac must have borne directly almost against the gullet; though, too, the wall of the sac may be gone at the spot, and portions of its fibrinous coagula protrude slightly into the interior of the œsophagus: Brader, U. C. H., Females. loc. cit.: I repeatedly saw this woman swallow with perfect ease. In King, too, U. C. H., Males, loc. cit., a smaller perforation existed without dysphagia.

1512. (*e*) Painful, hoarse, clanging, laryngeal cough, laryngeal honchi, dry or moist, audible sometimes at a distance, and yspnœa, at once habitual and increasing paroxysmally, coupled ith various morbid states of voice, indicate deep disturbance, unctional or organic, or both, of the larynx. The speaking oice may be husky, muffled, cracked, and hoarse; or simply eakened, or tremulous and variable in note, or actually lowered register. The hoarse variety appears to depend on chronic ryngitis, with diminished current of air,—itself, in turn, traceble to pressure on the trachea; pressure on a main bronchus is ot sufficient for the purpose. Œdema of the glottis, depending n congestion, venous or sub-inflammatory, has in some cases een found. Paralysis and atrophy of the muscles of one side f the larynx, coupled with flattening and compression of the ecurrent nerve, explained extreme vocal feebleness in a case bserved by Dr. Todd. Tremulousness and variation of note ve been traced to simple pressure on, and displacement of, e trachea. Paroxysmal dysphonia is explained by irritative action of the recurrent nerve. The trachea, chronically inmed, where the subject of irritative compression, is tender to the touch; and stridulous breathing is in part due to these conditions.

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* I have met with one case of repeated hæmorrhage through the mouth, ving all the characters of hæmatemesis, and in which, the lungs and stomach t being demonstrably unsound, there are several signs and symptoms of eurism of the aortic arch.

1513. Various forms of pain are, or may be, felt in the chest. First, immediately over the aneurismal prominence, pain may be produced by mere distension, by local pleurisy, or by irritation of the intercostal nerves. Secondly, pain in the neck and arms, down to the finger-ends, is traceable to irritation of the branches of the cervical and brachial plexuses. Thirdly, pain of a peculiar gnawing, terebrating character, constant, but increasing paroxysmally from time to time, exists at the dorsal spine, if the vertebræ are undergoing absorption. Fourthly, local pain and tenderness over the sternum, is sometimes connected with periosteitis going on to suppuration.* The second class of pains are of shooting, piercing, or stinging character, may be brought on by the slightest movement, even that of turning in bed, and are more or less paroxysmal. The gnawing pain in the back cannot, as some persons have supposed, depend on irritation of the roots of the spinal nerves, or it would, contrary to what is the fact, radiate in the course of their branches. The tenderness of an aneurismal prominence is sometimes extreme. Sometimes this is associated with a sensation of heat, perceptible also to the hand of the observer. In addition to all these sufferings, a feeling of fulness, weight, load, tightness, and oppression is experienced within the chest, coupled with a dread of movement, lest something should be displaced by the change of posture. There are patients who suffer seriously from spasmodic contractions of the diaphragm, or sensation of constriction round the base of the chest,—the obvious results of irritation of the phrenic nerve.

1514. The state of the respiration varies. If there be no pressure on the tubes, or irritation of these, or pressure or irritation of the vagi, recurrent, or pulmonary nerves, the breathing is calm. Where any of these conditions are present paroxysmally, the breathing undergoes temporary acceleration in proportion to their amount. Or, if they are permanent evils, the breathing is laboured, whistling, stridulous, audible at a distance, and the patient commonly points to the trachea, at the sternal notch, as the source of difficulty. The number of respirations per minute almost always exceeds the average of health more or less,—I have found it range from twenty-four to fifty-six. The pulse-respiration ratio is subject to great variation; thus, in one of the cases already referred to, the mean ratio throughout the time of observation being as 2·9 : 1, the extremes were as 5·3 : 1 and as 2·3 : 1; in this instance, the variations of broncho-laryngeal symptoms furnished a key to the rises and falls.* The chest-play is more or less confined. If a main bronchus be encroached on, or the mass of one lung diminished by pressure, the play on the corresponding side will be relatively deficient; and there may be special want of expiratory rather than of inspiratory power.† Dyspnœa is an almost invariable symptom where the sac is of any bulk, though all sensation of the kind may positively be wanting where the sac

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* Downie, U. C. H., Males, loc. cit.

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* Brader, U. C. H., Females, loc. cit. But this explanation will not always hold good: a ratio of 4·4:1 may coincide with very considerable irritation of the whole broncho-tracheal tract,—Mack, U. C. H., Males, vol. ix., p. 210.

† Thus, in Brader, the measurements of the chest below the fold of the mamma, gave:

	Medium.	Inspiration.	Expiration.
Right . . .	17⅛ . . .	17¼ . . .	17⅞
Left . .	16½ . .	16¾ . .	16½

Hence the total play on the right side equalled three-eighths, on the left two-eighths, of an inch. The left bronchus was "distorted, and almost obliterated by pressure of the sac." Hence it appears, too, that the excess of the right over the left side, at the end of full inspiration, was half an inch; at the end of full expiration, three-eighths of an inch. These results prove that there was a deficiency of expiration on the left side. They are corroborated by the facts, that the difference between full inspiration and full expiration on the right side equalled three-eighths of an inch, on the left only a quarter of an inch; while on the left side there was no difference, on the right a difference of a quarter of an inch, between the medium state and full expiration.

is even huge.* In many instances, the first symptom attracting the patient's attention, commonly increasing gradually in intensity, dyspnœa acquires, *cæteris paribus*, most intensity when the horizontal part of the arch is aneurismal. In addition to the pulmonary causes of the symptoms enumerated above, may be mentioned pressure on the auricles, pulmonary veins, or pulmonary artery, and passing accumulation of blood in the right cavities of the heart. Dyspnœa is prone, too, to occur paroxysmally at night, from accumulation of sputa, pressure on the trachea, produced by accidental movement into such positions as throw the sac against that tube, and probably from reflex action. The act of deglutition sometimes induces a severe fit of obstructed breathing.

1515. Cough, rarely absent, may be loud, dry, and paroxysmal,—paroxysms of the kind sometimes terminate in a syncopal state, or, after great effort, are relieved by expectoration of a thin, watery fluid. Unless under the influence of accidental inflammation of the air-tubes, there may be no expectoration at all. Blood may be discharged through the trachea in different manners. The sac and windpipe undergoing an extensive rent, a tremendous flow of blood may take place, and kill instantaneously; or, syncope occurring after copious discharge, coagula form and plug up the opening for the time;—upwards of a quart of blood may be poured out under these circumstances without immediately fatal result; but such temporary preservation of life is a rare exception to the common issue of such ruptures. Or, lastly, the expectoration may be habitually tinged with blood, so as to produce the red-currant jelly appearance: such expectoration, which is very unusual, does not always derive this character from blood filtrating from the sac, but sometimes from pressure on the vessels of the lung.† Moderate discharge of blood, by diminishing the size of the sac, and also by diminishing congestions, sometimes affords great temporary relief of symptoms.

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* Harris, U. C. H., Males, loc. cit.

† Brader's case, loc. cit., p. 121.

1516. Certain physical signs connected with the lungs are worth attention. Where the trachea is pressed on, the suprasternal region sinks in very deeply on inspiration; rhoncho-respiratory fremitus, in consequence of the powerful stridor of respiration, is carried to its maximum point. Pulmonary percussion-dulness comes of various influences exercised by the sac on the substance of the lung, displacement, condensation by pressure, and collapse from obliteration of bronchial tubes. Respiration-sound may be deficient to almost suppression, from bronchial pressure, through part or the whole of one lung, while exaggerated respiration exists elsewhere. Condensed strata of lung yield high-pitched bronchial respiration; and dry and moist bronchial rhonchi are audible [1297]. Jerking rhythm may sometimes be caused by the pulsation of a loose sac against the trachea or lung [1531].

1517. (*f*) The *heart* may become hypertrophous, especially if the sac originate near the sigmoid valves: but such effect is by no means constant; I have known the heart quite within the limits of healthy size under the circumstances. In some cases, the relative sizes of the arterial outlets of the heart undergo perversion; in one remarkable instance, the pulmonary orifice, when opened out, measured two and a quarter inches, while the aortic reached four and three quarters: this state of things must, in all probability, seriously increase dyspnœa.* Inequality of force and fulness of the radial, carotid or subclavian pulses, at corresponding points of the two sides, occasionally exists. Though other explanations have been suggested, it appears that pressure or obstruction with coagula at the aortic origin of the weakly beating vessel is the only positive cause of the difference.† The vessel on the affected, beats a little later, too, than on the sound, side. There is a certain sharpness and jerking character in the pulse, sometimes not unlike that of slight aortic regurgitation:—but the superficial arteries do not beat visibly. The pulse is sometimes bisferiens; however, observation does not justify the notion that the second wave depends on reaction of the aneurismal sac. The veins of the chest, of one or of both upper limbs may be enlarged, full, and knotty; sometimes so firm that they cannot be obliterated by pressure; those of the arm have been known to undergo complete obliteration by coagula. I have never seen pulsation of the jugulars in these cases. The situation of the obstructed veins will guide to that of pressure, whether on the superior cava or either innominate vein alone.

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* Downie, U. C. H., Males, loc. cit.

† Change in the pulses may, as might be anticipated, be pretty speedily

1518. (*g*) The bronchial glands are sometimes enlarged, and increase the percussion-dulness of the aneurism in the back.

1519. (*h*) The urine frequently contains excess of urea; it is free from albumen: on the whole it is, *quoad* diagnosis, insignificant.

1520. (*i*) The genital organs present nothing special.

1521. (*k*) Cephalalgia, a frequent symptom, sometimes depends on the throbbing action of the arteries, and is sometimes simulated by pain in the nerves of the scalp, from pressure on the plexuses below. Partial paralysis, sensory and motor, of an arm has occurred from pressure on the brachial plexus. The intellect is habitually unaffected to the last. It is quite conceivable that obstruction of the innominate or of the left subclavian, may be carried to such a point as to interfere with the nutrition of the brain and induce symptoms; but such result is, clinically, at least very rare.

effected. Thus, in Mack, U. C. H., loc. cit., p. 210,—"the left radial was feeble, yet sharpish, the right so excessively feeble that it was with difficulty counted." Dr. Reynolds had seen this patient one fortnight before, and was positive no difference then existed between the two pulses. Where difference of the kind exists, the arteries of the lower extremities should always be examined, lest a normal irregularity in the two sides be taken for the result of disease.

1522. (*l*) Paraplegia has in rare instances followed erosive destruction of the vertebræ and pressure on the spinal cord. In one remarkable case death occurring with sudden paraplegic symptoms and indications of rupture into the chest, the sac, it was discovered, had burst into the pleura, and also into the spinal canal.

1523. (*m*) As a rule the organs of sense escape—at least such amount of implication as readily attracts attention. I have known one pupil very notably smaller than the other, where no cerebral symptom of any kind had existed.*

1524. (*n*) In addition to the neuralgic pains in various nerves from pressure, absorption of their substance is sometimes effected. Intercostal neuritis has in a few instances been detected.

1525. *Course.*—In the great majority of cases the progress of aneurism is slow and insidious at first: the symptoms may, however, be suddenly developed, probably from some sudden increase in the bulk of the sac,—an increase generally connected with change from the simple to the mixed condition of the disease. The further course of cases is either gradually or interruptedly progressive: occasionally aneurisms of considerable dimensions have remained latent to the last, and their physical signs been merely those of a soft, solid mass within the chest. Physically considered, the course may be centripetal or centrifugal, or both combined: when aneurism combines both modes of progress, one almost always predominates in activity over the other.

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* Mack, U. C. H., Males, vol. ix., pp. 211, 244. "April 23. . . . . left pupil, in medium state, about $\frac{1}{8}$ inch in diameter; right not more than half the size; both are round and moderately brisk." This state held on during life, but after death—"May 21. . . . pupils round, both larger than during life; the right, that which was during life so notably the smaller, is now very distinctly the larger, of the two." In this case the innominate artery was so compressed that a narrow button-hole slit alone opened into the arch, and the right radial pulse was almost imperceptible; but the relative state of the carotid supplies cannot be appealed to in explanation of the unequal sizes of the pupils,—as Dr. Reynolds had seen them thus unequal at a time when he found the radial pulses equal: unfortunately the brain was not examined.

1526. *Mode of Death.*—There are five chief ways in which life may be destroyed in the subject of aneurism of the arch: by gradual asthenia and exhaustion; by the obstructive and irritative effects of pressure; by rupture and hæmorrhage; by acute intercurrent diseases; by co-existing chronic diseases.

(a) On the whole, actual death by gradual exhaustion is rare. Pain, insomnia, anorexia, wasting and low irritative fever, not unfrequently, it is true, bring the patient to the brink of the grave; but in several instances of the kind, observed by myself, the immediate cause of dissolution belonged to one of the other categories, especially rupture. In one melancholy case, the patient, unable to bear the anguish of intercostal and other neuralgic pains, which were fast undermining him, committed suicide [1531].

(b) Whether the aneurism affect the ascending or the transverse portions of the arch, broncho-tracheal pressure and irritation rank among the most frequent causes of death.

(c) Solution of continuity in the walls of the sac may be effected by slow perforation, or sudden rupture. Perforation, with or without oozing, or slight pouring out of blood, may occur into parts of considerable importance, not only without immediately fatal results, but without any perceptible special effect;* the occurrence of a sudden rent of any size completely through the wall of the sac, no matter with what organ or part an unnatural communication be thus set up, is however almost invariably fatal at once. There is no conceivable position into which fatal rupture has not occurred, as may be seen by examination of Dr. Crisp's valuable collection of recorded cases. Where the ascending part of the arch has been affected, the pericardium has proved the most frequent seat of hæmorrhagic effusion. Of 138 cases, where Dr. Crisp found the mode of death stated, six only furnished examples of external rupture.

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* Brader, U. C. H., loc. cit., into œsophagus. Whittaker, U. C. H., Females, vol. vi., p. 174, admitted immediately after violent hæmorrhage, having the characters of hæmatemesis, rallied, had three more attacks within thirty-six hours, dying instantaneously in the last. The stomach contained fluid blood 7 oz., and two massy coagula weighing 13½ and 7 oz.; the sac, occupying the descending portion of the arch, communicated with the œsophagus, by an opening as large as half-a-crown.

Hæmorrhagic effusion from a sac may be retained, as when it pours into the pericardium, pleura, mediastina, spinal canal, pulmonary artery, vena cava, or various parts of the heart; it is rejected, when poured into the trachea, a bronchus, the lung-substance, the œsophagus, or through the chest-wall. Large amounts of blood may be discharged, not only without deleterious effect, but actually with beneficial influence, through relief of congestion, and diminution of various irritative pressures.

Death may also take place by rupture of the heart [1448], or of the aorta itself on the cardiac side of the aneurism.

(d) Death by dependent acute intercurrent diseases, such as pneumonia, gangrene of the lung, bronchitis, pleurisy, pericarditis, is occasionally observed. Death through independent acute intercurrent diseases is very rare; the victims of cholera asiatica, continued fever, rheumatic fever, the exanthemata, acute Bright's disease, cerebral and abdominal inflammations, are scarcely ever the subjects of aortic aneurism.

(e) Of co-existing chronic diseases, affections of the substance and valves of the heart, preceding, coincident with, or sequential to the arterial changes, are the most frequently conducive to the fatal issue: a rare example of death from phthisis now and again occurs; moderate emphysema is not very uncommon. Diathetic diseases of all varieties are rare; aneurism attacks people of the most vigorous frame of constitution.

1527. *Repulsions and Affinities.*—In 108 cases of aneurism, Rokitansky found tubercle only five times—and always retrograde tubercle. Twelve cases of aneurism of the arch, by Dr. Greene, supply four examples of tubercle; 132 cases of aneurism of the arch, collected by Dr. Crisp, furnish two examples of death by phthisis. Dr. Stokes teaches, on the other hand, that "the morbid condition which most often accompanies aneurism is tubercle. . . . I have often thought that there was a case deserving the name of consumptive or strumous aneurism, in which the same general morbid state, which caused deposition of tubercle in the lung, simultaneously affected the coats of the aorta." According, then, to Rokitansky, tubercle and aneurism are absolutely antagonistic; according to Dr. Stokes, of affinity so close that, in some cases at least, one and the same diathesis simultaneously generates both.

In fourteen cases of fatal aneurism of the arch, examined by myself post-mortem, one rather doubtful example of tubercle occurs, in a male aged fifty-one; in one other male, aged thirty-two, there were pulmonary cavities, and growing tubercle; in all the other cases, the absence of crude tubercle or grey granulation is affirmed in my notes. On the other hand, I do not remember, in the vast number of phthisical people I have opened, to have met with any instance of progressive aneurism. Hence my contingent of experience supports the inference of Rokitansky as to the rare association of the two diseases. But the disparity of the ages most apt for the generation of tubercle and aneurism must not be forgotten; the mean age in my cases was forty-two; and it may be calculated that, omitting children altogether, about 63 per 100 of tuberculous adults, 13·5 only per 100 of aneurismal people, are under thirty years of age. The syphilitic cachexia and gouty diathesis are said to have some connection with aneurism: the fatty diathesis has possibly a stronger claim to the character.

A parallel, which I cannot help thinking somewhat fanciful, has been drawn between aneurism and cancer. Like the cancerous, it is said, the "aneurismal diathesis" is never extinguished: what comes, then, of the cures, spontaneous and by art, of the disease? Frequently, we are assured, many of the arteries are involved in the same person; the assurance is directly at variance with statistical returns: besides, admitting the fact, the analogy fails; cancers multiply through the blood; aneurism could only do so through local changes in the vessels. The aspect of the patient and the general decay of the organism resemble those observed in cancer, according to this argument: the statement is decidedly inapplicable to the majority of cases of aneurism, and only true of those attended with an extra share of suffering. Besides, in their mode of distribution to the two sexes, aneurism and cancer are well nigh the antipodes of each other.

1528. *Diagnosis.*—Locally considered, the diagnosis of sacculated aneurism of the arch turns essentially on the co-existence of *a pulsating prominence,* visible and palpable, limited in area, and corresponding in seat to that portion of the vessel; the *signs of internal pressure; certain arterial murmurs and sounds;* and *limited percussion-dulness in the course of the arch.* But a pulsating prominence may also be produced by a small solid mass lying over and receiving the impulse of a sound aorta,—the pulsation of a mass of the sort may even be quasi-expansile; or it may be due to an abscess in the mediastinum; or to pulsating empyema; or to a tumour pulsating interstitially. Secondly, the signs of internal pressure may be produced by solid tumour. Thirdly, a double hoarse murmur over the arch, inaudible or only faintly audible at the heart,—a diastolic murmur similarly localised, and a pumping or sucking character of the aortic sounds, without murmur, are the most significant auscultatory signs. Fourthly, several morbid states of the mediastina, bronchial glands, lungs, pleura, and even of the chest-wall, may accidentally render the percussion-sound dull in the course of the arch.

But the true difficulty in the diagnosis arises, when not one of these physical signs exists with satisfactory distinctness; and when, in point of fact, the nature of the disease must be elicited on indirect evidence, and *per viam exclusionis.* The absence of symptoms and signs, indicative of ordinary affections of the heart and lungs, in an individual suffering from persistent anomalous disturbances within the chest, even though he does not, or rather because he does not, exhibit any failure of general health, affords strong motive for suspecting aneurism. If under such circumstances copious hæmoptysis occur, the diagnosis of aneurism, though undefended by a single positive physical sign, would rarely be at fault.

Tuberculous consolidation of one apex, especially the left, with murmur in the subclavian or pulmonary artery, is distinguished from aneurism by the non-extension of percussion-dulness across the middle line; by its extension, on the contrary, to the acromial angle; by the existence of some tone in the percussion-sound, and some resilience in the wall of the chest; and by the absence of pressure signs, eccentric or concentric. The symptoms, local and general, are also different.

Fluid in the pericardium is distinguished by the pyramidal form of its dulness, which aneurism never simulates except under the very rare accidental circumstances already referred to [1198]. In a case of *enlarged heart*, there is but one centre of motion—in aneurism two; itself and the heart. If the sac be quiescent and non-expansile, however, the site of the percussion-dulness and the existence of pressure-signs must be appealed to for the distinction on the side of aneurism, and more or less of its special signs sought for on the side of enlarged heart. If general dropsy exist, this is in favour of heart-disease, positively against aneurism as the sole disease.

Pulsating empyema, with its throbbing prominence near the edge of the upper bone of the sternum, simulates aneurism closely, but may be distinguished by the rules elsewhere set down [729].

A chronic sub-periosteal abscess of the sternum, forming a small prominence in the line of the transverse portion of the arch, fell under my notice some time ago,* sufficiently resembling an aneurismal sac. But there was no impulse; gentle percussion immediately round the prominence gave clear resonance; there was no murmur, and concentric pressure-signs were totally absent. But there might accidentally have been impulsive action of the vessel beneath, and excess of mediastinal fat might have rendered the percussion-sound dull; under such circumstances the diagnosis would have been excessively difficult.

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* Marg. Mottlee, U. C. H., Females, vol. v., p. 56.

Infiltrated cancer of the lung causes retraction of the side, produces no local prominence, deepens the intercostal spaces, and frequently renders the percussion-sound tubular in the infra-clavicular region; it does not produce pressure-signs, and may be the seat of the signs of softening and excavation.

Tumour in the anterior mediastinum presents the greatest number of positive points of similarity to aneurism. Now, if there be highly-marked pulsation, a broad-based prominence with conical elevation in the centre, the murmurs most distinctive of aneurism, and a sensation of the flow of liquid beneath the integuments, there can be no doubt that, whatever other grounds for diagnosing tumour may exist, aneurism is really present. But every one of these things may in cases of aneurism be absent: then, observe how like the two things are —a sac filled with fibrine and a solid tumour. In truth, one is a tumour *inside*, the other *outside* the arch: and obstruction from without may have the same effect as from within on its circulation. Common to the two things are dulness and non-resilience, usually extending across the middle line, all the signs of centripetal and all the signs of centrifugal pressure. Under such circumstances, the question becomes one of pure probabilities. The conditions in favour of aneurism would be these: situation in the course of the arch, vibratile thrill above or below the clavicle, gradually increasing nearness of pulsation to the surface,* dysphagia, great pain, especially of the dorsal spine, absence of œdema of the arm and chest. The circumstances in favour of tumour, and against aneurism, would be the facts of the patient being a female* and under twenty-five years of age; great superficial extent of percussion-dulness, especially if there were no marked attenuation of the walls of the chest; absence of any heaving motion in the affected spot; want of accordance between the sites of maximum-dulness and of pulsation; and currant-jelly expectoration common with tumour, very rare with aneurism.

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* But, from stratification of fibrine, the pulsation of an aneurism may grow

It is a curious fact, that where a *quiescent aneurismal sac* and a *tumour* co-exist, the usually essential sign of aneurism, namely pulsation, apparently expansile, may be furnished by the tumour; and the usually essential sign of tumour, dead, pulseless dulness under percussion, may be caused by the aneurism. This statement is well illustrated by the case of Brader (loc. cit.)

The means of distinguishing *coarctation of the arch* have already been examined [1485-6].

Aneurism of the innominate artery is distinguished by the higher position of its pulsating prominence behind or above the inner part of the clavicle; prominence appears relatively early; dysphagia, tracheal pressure-symptoms and dyspnœa are comparatively rare; the clavicle is often pushed from its place; paralytic symptoms in the right arm are greatly more frequent; the respiration-sounds are seldom enfeebled,—if they are so, the right lung suffers. In an elaborate essay by Dr. Holland† I find it stated, that the arteries in the right side of the neck and head and in the right arm generally pulsate less strongly than on the other side,—whereas the reverse is the general fact in cases of aneurism of the transverse portion of the arch.‡ The

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deeper, and, on the other hand, that of a tumour may become more superficial.

* But this is of little value, for the excess of aneurism of the arch in males is by no means so great as that of aneurism of all arteries indiscriminately.

† Dublin Journal, 1852.

‡ This rule is, however, open to numerous exceptions; in my own cases of

same writer notes that pressure on the right subclavian and carotid diminishes or stops the pulsations of an innominate sac, —while, brought to bear on either the right or left arteries, it exercises no influence on aortic aneurismal action.

1529. Group *b.—Fusiform dilating aneurisms.* The peculiarities of these aneurisms are, as compared with the sacculated, diffuseness of pulsation above and below the clavicle, visible and palpable, comparatively much less below than above, though even there, if anæmia exist, it may be very considerable; more thrill above, less below, those bones; rough, prolonged, rasping, whizzing or whirring murmur, systolic only, audible along the arch, and louder there than at the aortic valves, if they also be the seat of murmur. Centripetal and centrifugal pressure-signs are almost or completely wanting.

1530. Anæmia and nervous excitement may simulate such dilatation; the results of percussion will distinguish the cases.

1531. Examples of *globular dilatation of the arch* are so rare, that a note of the principal phenomena, observed in the only case I have seen, will not be misplaced:—

Mr. ——, æt. 50, January 13, 1852; intense pain, upper sternal and left nfra-clavicular region, radiating to axilla and elsewhere variously; no murmur ver heart, nor aorta in front; distinct systolic short superficial weak murmur n course of thoracic aorta, just at the level of the inferior angle of the capula; no prominence or impulse visible in infra-clavicular region; no thrill bove or below the clavicles; percussion dull in left inter-scapular region, nd slightly so even in the right, also about first and second left costal cartilages n front, but patient so sensitive that careful percussion impossible. Respiration left inter-scapular region, and below this, peculiarly jerking,* of very sharp owing quality. March 31; impulse now visible, as also very slight general rominence about the top of the sternum, and the two upper left costal cartiges; no thrill; no murmur in heart or aneurism anteriorly; double sound ver prominence, both divisions louder than at the heart. April 26; promi-

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cending and transverse aortic aneurism combined, the weakly acting radial is more frequently the right.

* The sac, being exceedingly loose, probably caused the jerking rhythm of e respiration by pulsating against the main bronchus.

nence more obvious, and area of dulness increased ; absence of thrill or murmur as previously.

This patient destroyed himself a few days after the last interview, all the measures employed failing to relieve his pain. The aneurismal dilatation was of large size (U. C. Mus., No. 4036).

1532. Group *c.—Mixed aneurism.* The sudden extension of dulness in the situation of a simple sac, coupled with similar increase of pressure-signs, especially if these conditions follow effort of any kind, makes it probable that the inner and middle coats have given way, and the outer undergone additional pouching. But in the signs of a mixed aneurism, when actually developed, there is nothing special. The prognosis is rendered worse by the yielding of the inner coats.

II.—ANEURISM OF THE DESCENDING AORTA.

1533. The signs of an aneurism seated between the termination of the arch of the aorta and the diaphragm will, of course, vary somewhat with the precise portion of the vessel affected. If the sac be not of large dimensions, little is to be learned by inspection ; however, in certain positions, in consequence of its lying behind the heart and pushing this organ directly forwards against the ribs or sidewards, the maximum cardiac impulse may be transferred from the apex to the base, and so-called diastolic impulse also produced. Posteriorly the hand may detect slight arching to the left of the spine; if the arched surface be the seat of the least impulsive action, the sign becomes one of importance. Dulness under percussion, limited to the same situation, and inexplicable by the condition of the lung, heart or pleura, would, of course, strengthen the inference drawn from the previous sources. An aneurism in this situation may supply the varieties of murmur and sound already enumerated; murmurs must be stronger over the sac than over the heart, to have any diagnostic value. Feebleness or deficiency of respiration close to the spine, or over the side generally from pressure upon the main bronchus, will corroborate the other signs.

1534. Vertebral gnawing and intercostal neuralgic pains, coupled with sensation of internal throbbing action, sometimes with difficulty distinguished from that of the heart, are observed in these cases. The laryngeal system, generally saved by its distance, sometimes suffers through extension of irritation from a main bronchus; the trachea, indeed, has, in rare instances, undergone direct pressure from very large-sized sacs. Perforation of the main bronchus, dysphagia from mechanical obstruction of the œsophagus, or, where the disease occupies the immediate vicinity of the cardia, various gastric symptoms simulating obstruction of that orifice of the stomach, have been noticed.

1535. Death occurs more frequently, perhaps, from rupture into the œsophagus than from any other single cause; curiously enough, rupture into the right, occurs almost, if not quite, as frequently as into the left, pleura. The patient dies, worn out by the effects of bronchial and tracheal pressure in some cases. Aneurism of this division of the aorta is singularly rare in females.

1536. The affection with which an aneurism, thus seated, may most readily be confounded, is hypertrophy of the heart; the strong systolic impulse, and the diastolic impulse combine to deceive. But careful employment of all the methods of physical diagnosis will prevent error in well-marked cases; while it must be confessed that very small sacs behind the heart, unless some accidental circumstances throw light on their existence, are exceedingly difficult of positive detection.

1537. *Duration and prognosis.*—There is an unfortunate deficiency of materials on any large scale for determining the mean clinical duration of the disease. That, once developed, it will eventually destroy life is an inference practically unshaken by the few recorded examples of alleged cure. But it is astonishing how long life may be prolonged, even with tolerable comfort to the individual, by such simple management, hygienic and medicinal, as common sense dictates. I shall not easily forget the case of a man first seen in November 1846, with a large aneurismal prominence, pulsating so liquidly, if I may use the word, and so directly under the skin, that it became necessary to apply a mechanical protection against the danger of rupture from a blow or even from sharp friction,—a man worn with suffering,—unable to sit, lie or stand, from pain and general uneasiness,—and wasted considerably in flesh and strength: and yet the death of this patient did not take place till July 1849,—he having, meanwhile, though actively phthisical also, lost the major part of his more serious sufferings, and, in a certain subdued fashion, actually enjoyed existence.*

It is positive that the mean duration is greater, when the sac grows centrifugally than centripetally.

1538. *Treatment.*—In proceeding to the treatment of an aneurism of the thoracic aorta, the first point is to determine, if possible, whether we have a fusiform or globular dilating aneurism, or one of the sacculated varieties, to deal with. For, whilst coagulation of the blood within the aneurism is scarcely obtainable in the former cases, and if obtained will not effect their cure; in the latter, there is a natural tendency to such coagulation, and coagulation does actually promote anatomical cure. Hence it appears that the clinical distinction of these kinds of aneurism is not a piece of mere scholastic refinement, as it has been slightingly called, but a matter of practical importance.

There is another point worthy of serious consideration. With what hope of achieving a cure do we undertake the treatment of the case? If, as experience amply shows, existing methods have seldom, if ever, accomplished removal of the disease, clinically understood, the slightest risk should obviously be avoided of doing mischief to the constitution by the energetic employment of any one of those methods.

1539. (*a*) In cases of fusiform and globular dilatation, the indications are the prevention of enlargement and rupture of the aneurism. These ends may be best secured by occasional leeching over the affected part, especially if there be local tenderness, —much better than by small bleedings from the arm. Full and repeated venesection, on the plan of Valsalva, is in this species of aneurism even less permissible than in the sacculated varieties: but a single abstraction of some eight or ten ounces of blood at the commencement of treatment, more especially in plethoric persons, sometimes gives considerable relief to distressing symptoms within the thorax,—even this should be cautiously done however; if the system at large has at all suffered from the disease. Purgatives, to such amount as to maintain a tolerably constant free action from the bowels, both from their sedative effect on the circulation, and from their preventing the necessity for effort in defecation, are essential. Diuretics do service by preventing the water in the blood from rising above par. Direct sedatives of cardio-vascular action, digitalis, aconite, hydrocyanic acid, and belladonna, internally and externally, lessen the violent pulsatile action of the diseased vessel, and may be given in various combinations, and more or less steadily.

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* Harris, U. C. H., Males., vols. ii. and viii.

The diet should be so arranged as to support, without exciting or over-nourishing: but anæmia is even more baneful than plethora, and the starvation system must be studiously avoided. The patient should abstain from all excitement, mental and emotional, pass the greater part of his time in perfect rest, and take but moderate daily exercise on foot: carriage exercise on smooth ground may be permitted to any amount desired.

1540. (*b*) In the instance of a sacculated aneurism, the object being to promote coagulation, occasional venesection, with the view of lessening the force of the current, is commonly recommended. But there is a double danger to avoid here: if too much blood be drawn, the action of the circulating system will be excited, instead of tranquillised; and if the quality of the blood be seriously impoverished, the softness of the coagulum will probably render it comparatively useless as a support to the distended walls of the vessel. Theory, in truth, does not, at the present day, support the ideas of Valsalva concerning abundant depletion; and since the diagnosis of aneurism has become somewhat positive, cures obtained by his system have ceased to be heard of. True, the recommendations of Valsalva are rarely, if ever, executed to the letter: the courage of patient and physician generally fails—in time, probably, to prevent irremediable mischief. Moderate leeching over the sac from time to time is always useful. Digitalis and other cardiac sedatives promote coagulation by enfeebling and slackening the current; and if there be no contra-indication in the state of the heart, a fair trial of them should never be omitted. Some practitioners have much confidence in the acetate of lead. Purgatives and diuretics are useful on the principles, a moment since referred to; the latter especially because, while they diminish the water, they exercise no influence on the fibrine, of the blood.

Cold poultices of linseed meal and vinegar, or of conium and digitalis, relieve local suffering, and probably promote coagulation. Ice to the surface is grateful to some patients, unbearable by others: it can rarely be kept applied for a sufficient length of time to modify the circulation beneath. Cold poultices of oak bark have appeared to me useful. If there be excessive pain over the sac, the application of the freezing mixture, as recommended for anæsthetic purposes generally by Dr. James Arnott, would, for a double reason, deserve a cautious trial. The local application of chloroform I have also found useful. Counter-irritation in the neighbourhood of, but not over, the sac sometimes relieves greatly. I have known interscapular pain completely disappear under the use of caustic iodine.

Tannic and gallic acids, combined with digitalis, aconite, or belladonna, have appeared to me to exercise a very beneficial effect in promoting coagulation: gallic acid may be given in doses of three, five, and eight grains twice or thrice daily, with occasional intermissions, for a length of time,—its constipating effects being obviated by occasional doses of castor oil.

The theoretical necessity for fibrine of good quality being clear in these cases, such diet is advisable as seems to promote its formation. However, the dangers of plethora, especially as much exercise cannot be permitted,* must be held constantly in view, and over-nourishment avoided. Fluid in any quantity is injurious, and stimulants seriously baneful.

Galvano-puncture, as originally suggested by Pravaz for external aneurisms, has occasionally been thoroughly successful, even with vessels of considerable calibre,—as the external iliac†: but occasionally the results have been disastrous,—and the application of the plan to the aorta would be so imminently hazardous in various ways as to be unjustifiable. The same may be said of the injection of a few drops of the perchloride of iron; but the free administration of the salt internally, as prepared by Burin for M. Pravaz, seems worthy trial.

1541. If the aneurismal sac be very superficial and its wall ill-protected by fibrine near the parietes of the chest, it may be necessary to apply a shield, fitted to the part, to protect it from the chances of external violence.

The laryngeal symptoms of aortic aneurism have frequently been mistaken for evidences of disease in the windpipe itself, and tracheotomy vainly performed for their relief. Dr. Gairdner, however, endeavours to show that the operation presents a fair claim to be admitted into the legitimate treatment of aortic aneurism,—"not unwillingly and as a last resource, but as early as it could be ascertained that laryngeal symptoms were the source of the more immediate danger." I have had no personal experience of the operation in cases of aortic aneurism; but can conceive it might become justifiable as a means of *probably* prolonging life for a few hours, where such prolongation might be a matter of importance.*

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* No argument in favour of exercise appears to me to flow from those exceptional cases, in which violent physical exertion is borne, at the moment, with impunity. Thus it is well known that the celebrated and lamented surgeon, lately carried off from among us by aneurism of the transverse portion of the arch, some time after he had lost quarts of blood from the sac, and while already seriously distressed by tracheal pressure-symptoms, performed feats of personal prowess, pedestrian and other, not only without immediate ill-results, but, as he himself maintained, with relief to his sufferings. The ultimate and real effect of such strain upon the circulation must have been to hasten the enlargement of the sac.

† Eyre, in Lancet, July, 1853.

Arguing from the "extraordinary relief" to symptoms often afforded, when an enlarging sac, compressed by the clavicle, at length succeeds in partially dislocating the bone forwards, Dr. Stokes suggests that, where such displacement of the bone failed to take place spontaneously, division of the sterno-clavicular ligaments might be effected with the knife.

1542. In not a few cases of aortic aneurism the treatment becomes less that of the disease itself, than of some associated state, as hypertrophy, or flabby weakness of the heart, or anæmia, —or of some sequential affection, such as obstinate bronchitis. Tartarized antimony, nitre, and digitalis in combination, are the best remedies for the latter malady.

ANEURISM OF THE ABDOMINAL AORTA.

1543. *Physical signs.*—Inspection may, or may not, disclose some abnormal appearance. If the sac be small, and especially if it spring from the posterior aspect of the vessel, or if the aneurism be of the fusiform species and not bulky in any part of its extent, the eye may fail to detect any peculiarity in the form of the abdomen. If the sac have acquired any size, pulsating prominence, of variable extent, is seen anteriorly, in the course mainly of the aorta, from the epigastrium downwards,* or bearing especially to the left side of the abdomen, in rare instances to the right. The surface is smooth to the eye; the respiration-movements laterally, or bilaterally, impeded.

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* A case recorded by Mr. Judd seems to show that life may by possibility be prolonged for several days, nearly a fortnight, by tracheotomy,—though the trachea suffers very serious pressure from a large sac: but obviously, we have no right to expect the repetition of so exceptional an occurrence (Lancet, 1844).

The hand, placed on the anterior prominence, receives a single systolic impulse, sometimes of enormous force and quite out of proportion with the volume of the moving mass, while posteriorly, in the lumbar region, no trace of impulse may be perceptible. Generally speaking, if the abdominal walls be thin, the hands may be passed on either side of the sac, and an estimate formed of its bulk: the impulse is felt to be laterally, as well as anteriorly, expansile. The chief pulsation may be to the right of the spine, the sac sometimes mainly growing in that direction. The mass, fusiform, rounded, smooth or lobulated, is immovable,† and commonly compressible more or less: caution is, however, requisite in ascertaining these particulars. In comparatively rare instances a second impulsive, or at least strongly jogging, action, diastolic in time, may be felt: or, on the other hand, pulsation may be absent when the sac is quite large enough to alter the form of the abdomen slightly; while, *per contra*, a very small sac sometimes furnishes powerful impulse. Thrill, systolic in the majority of cases, of diastolic time in rare instances, may be felt.

The left semi-circumference of the abdomen may, or may not, be increased.

The size of the sac can only, if at all, be accurately estimated by percussion; and the tenderness of the surface and the neighbouring parts generally often interferes with the process. Percussion of any force is dangerous and unjustifiable,—especially as intestinal note generally interferes with precise limitation. Besides, a considerable time before death, the apparent size of the sac may be greatly increased by successive and repeated extravasations of blood behind the peritonæum.*

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* Hallington, U. C. H., Males, vol. viii., p. 61. The visible prominence here extended from one inch to the right of the umbilicus, to the anterior spine of the left ileum: dilatation of the aorta commencing an inch below the origin of the renal arteries; sacculation implicating the lower part of the aorta and the common iliac.

† In rare instances the mass is moveable, and in such cases, Dr. Stokes points out, pulsation and even murmur may be made to appear and disappear.

I have heard in connection with aneurism thus seated: 1. A single systolic murmur, without sound of any other kind; 2. A dull muffled systolic sound, convertible into a murmur by a little pressure; 3. A sharp, abrupt, short systolic murmur at the left lumbar spine, much more marked than in front; 4. A systolic murmur below the sac, none immediately over it; 5. Occasionally a dull second sound. I have never heard a murmur diastolic in time. In some instances systolic murmur is audible in the reclining, when inaudible in the erect, posture: Dr. Corrigan supposes this explicable by the removal of hydrostatic pressure in the former position, and the consequent greater freedom of the current in and out of the sac; but systolic murmur may be totally absent in every possible posture. The characters of murmur vary as in the arch of the vessel: its amount sometimes changes inversely as the growth of the aneurism. It may be so loud as to be audible at a little distance from the surface.†

1544. It is all-important for the observer of an obscure abdominal disease to remember that even large-sized aneurism of the ventral aorta may exist in the total absence of all positive physical signs,—neither impulse, murmur, nor percussion-dulness being discoverable: the subjective symptoms are then very likely to deceive.

1545. *Symptoms.*—The patient may or may not be conscious of pulsation, and this whether there be or be not, objective evidence of pulsation. Subjective pulsation, absent for two years, in a case observed by myself, then suddenly came on after effort in running, and continued until death. Pain following the course of nerves implicated by pressure—passing along the edge of the ileum down the thigh to the testicles and pudenda generally,—and in character raw, sore, pricking, cord-like, plunging, hot and burning at one time, cold at another, accompanied with spasmodic difficulty in passing urine, and with tonic contraction of the flexor muscles and inability to straighten the limb, the whole attended also with peculiar gnawing vertebral pain, existed in the case already more than once referred to. But obviously the neuralgic sufferings must vary with the exact site of the sac; sometimes they are relieved by special changes of posture. Theoretically, anasarca of the lower limbs, or of one of them, must occur, according as the inferior cava or either iliac vein is pressed on; but in practice either effect is most rare,—and fulness of the subcutaneous abdominal veins from obstruction of the cava may exist without any pedal œdema even. Wasting of the testicle I have seen from obliteration of the spermatic artery. Pressure on the descending or transverse colon may obstruct the bowels, and cause flatulence, constipation and great labour in defecation. In the case of Hallington (loc. cit. p. 64) the sac was adherent to the descending colon by pseudo-cellular bands, and the calibre of the bowel in one spot much reduced by contraction of these bands. Ascites of any clinically important amount is excessively rare, if indeed it ever occur through the influence of aneurism alone. The rarity of dropsy of any kind is in regard of diagnosis seriously significant.

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* The extravasated blood may make its way to the front of the abdomen, and extend upwards to the pleura. Hallington, U. C. H., loc. cit. The extravasated coagula may pulsate distinctly under the influence of the sac, and with expansile character.

† Case by Dr. Reynolds, "Med. Times," Sept. 1852, p. 284.

The respiration, if the sac be of moderate size and low down, s of natural frequency and character; when high or of notable bulk it interferes with phrenic action, throws the onus on the upper ribs, and accelerates the act somewhat.

The pulse, even while the patient is visibly perishing from pain, insomnia and exhaustion, need not be accelerated,—ranging from 78 to 88: the pulse-respiration ratio may be a normal one of 4·35 : 1. The heart may be raised upwards or pushed sidewards.

The urine may be rendered albuminous by renal congestion induced by pressure on the emulgent vein; otherwise it is perfectly natural, as far as the aneurism is directly concerned; but when the constitution begins to sympathise, I have observed continuous oxaluria.

1546. *Duration, and mode of death.*—In some instances these sacs have acquired enormous bulk: one preserved in the Fort Pitt Museum is said to have contained ten pounds weight of coagula. Hence the inference that the gradual growth of the disease is not incompatible with existence. But we have little information as to the mean duration of life after the outbreak of symptoms: I have known twenty-four, thirty, and thirty-eight months intervene between the earliest indications and the fatal termination.

Death occurs by rupture of the sac behind or into the peritonæum, into the pleura, lung, colon, renal pelvis, or mediastinum; or without rupture by jaundice, gangrene, exhaustion, &c.

1547. *Diagnosis.*—The diagnosis may be conveniently considered under the three heads of cases: (*a*) where there are abdominal physical signs; (*b*) where there are no physical signs, but serious ventral symptoms, mainly neurotic, with or without much constitutional sympathy; (*c*) where there are no signs, or very trifling ones, and no symptoms of the least apparent consequence.

(*a*) The difficulties sometimes arising in the distinction of mere *aortic pulsation* from aneurism have already been considered. [1472]. Curiously enough, the only case in which I have heard abdominal aortic murmur, diastolic in time, was one of diminished, instead of increased, calibre of the vessel. *Fæcal accumulation* is distinguished generally by the oval outline of the fulness; by its doughy inelastic feel; by the existence of several spots of dull and clear resonance under percussion close to each other, and within the area of the swelling, from the intermixture of gas with solidified fæces; sometimes from the position of the mass; and, generally, from the history of the case. The pains of aneurism may be imperfectly imitated by those of peritoneal distension from the enlargement of the bowel; but it is rare indeed that a mass of fæces receives such arterial impulse from behind as to simulate that of aneurism.—In the obscurity of their early symptoms, in the eventual pain, and in the gradual exhaustion they produce, there is considerable similarity between *lumbar and psoas abscesses* and aneurism; but the swelling of these abscesses passes in an elongated form from above downwards, and does not exhibit an irregularly globular shape, as aneurism sometimes does; they give neither impulse nor murmur. Tenderness exists in the lumbar spine, and there may be loss of motor power in the lower extremities; but the actual pain is materially less, as a rule, than in the aortic disease. Tubercles should be sought for in the lungs: their presence would be directly in favour of lumbar abscess, of tubercular origin,—against aneurism.—*Hydronephrosis and pyelitic distension* are accompanied with renal symptoms, changes in the urine, tumour with the characters of renal enlargement,—a tumour of tuberous nodular outline, non-impulsive, murmurless, and extending further into the flank and into the back than aneurism. The urine may be albuminous in all three affections.—*Tumours* of various kinds in the abdomen may pulsate in expansile manner, and be the seat of murmur; the murmur is high-pitched, whiffing invariably as far as I have heard; but a careful consideration of the whole case is the best safeguard against error. In a case of cancerous lumbar glands, seen some time since, not only were there expansile impulse and murmur, but such neuralgic pain as commonly accompanies aneurism; still the knowledge that a cancerous sarcocele had previously been removed prevented mistake.—An *enlarged lumbar vertebra*, pressing forward the aorta, will cause extra pulsation; but there is no lateral expansion of the vessel; and the murmur is whiffing or rasping, and not heard laterally.*

But even if there be positive surety of the existence of abdominal aneurism, we are not at once entitled to pronounce it aortic; it may be situated in the cœliac axis, the hepatic, the superior mesenteric, or the renal arteries. But the signs of these aneurisms have as yet been imperfectly investigated,† and I know nothing of any one of them by experience.

(*b*) Wherever obstinate abdominal neuralgic pains exist, especially in a male, and where the ordinary signs of visceral disease cannot be established, aneurism should be held in view as most probably present, even though there be no single physical sign to warrant such an opinion. Let the examination never be considered complete, however, without careful auscultation in the left vertebral groove. It will be necessary, too, to exclude, with as much certainty as possible, the presence of cancerous lumbar glands. An aneurismal patient of this class may be seen at a time when his general health is excellent, or when it has already failed.

(*c*) Slight pain in the lumbar region may be the sole symptom for twelve months, to my own knowledge, after aneurismal distension has probably commenced. Hence the importance of thoroughly investigating physically cases of alleged incurable lumbago and sciatica. Relief of lumbar pain by cupping will not disprove its aneurismal origin.

1548. The treatment is the same as of intra-thoracic aneurisms in general. Were the disease diagnosticated at an early period, might any good be effected by pressure either on, above, or below the sac?—a cautious trial of one or other form of pressure might with propriety be made.

1549. Group *d. Dissecting Aneurisms.*—The morbid anatomy of dissecting aneurism of the aorta, in its three essential varieties, is clearly demonstrable from existing records: its clinical history has yet to be worked out. And, indeed, from the nature of things it seems singularly unlikely that any general account, applicable even to the majority of such cases, can be given,—seeing that the symptoms must in great part depend upon the extent and precise portion of the aorta affected.

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* Vide case of Dr. Taylor, in Author's work on Cancer, p. 528.

† Vide Ballard, Physical Diagnosis of Dis. of the Abdomen, p. 217.

1550. The symptoms in recorded cases may clearly be referred to three heads, which the observer should always aim at severally distinguishing;—namely, (1.) symptoms of shock to the system at large; (2.) of dynamic and statical disturbance of the injured artery; and (3.) of mechanical interference with the function of organs supplied by branches from the injured part of the vessel. (1.) The symptoms of shock are, primarily, sudden faintness or actual syncope, and, on recovery of consciousness, nausea, vomiting, and pain in the thorax or abdomen: secondarily, febrile action, by no means necessarily very marked, thirst, furred tongue, abdominal tympanitis. (2.) The dynamic disturbances of the artery are signified by more or less severe pain in its course, and throbbing action, irregular in force and rhythm. Statically, the vessel and its injured branches are widened and rendered uneven; while the obstruction to the current, offered by the prominent and ragged lining membrane in the site of its ruptures, gives rise to blowing systolic murmur, which, if seated near the heart, may be mistaken for that of constrictive disease of the aortic orifice. (3.) The symptoms of mechanical origin are produced by the accumulation of the blood, filtrated between the coats of the aorta, against the orifices of arterial branches, whereby these are completely, or almost completely, blocked up. The nature of these symptoms will, of course, depend on the distribution of the blocked-up vessels. Thus, in a remarkable case, observed by Dr. Todd (Med. Chir. Trans., vol. xxvii.), where the innominate and the renal arteries were mainly obstructed, very singular cerebral symptoms and suppression of urine marked the event. If a main bronchus were pressed on by the suddenly enlarged vessel, equally sudden deficiency of breathing with clear percussion-sound, would ensue.

1551. Were the practitioner fortunate enough (guided by the sudden supervention of symptoms of the three classes now distinguished, and of a strong arterial murmur in a person known to have previously been free from this physical sign,) to divine the occurrence of acute separation of the coats of the aorta, it does not appear that, in the present state of knowledge, the treatment would be materially improved by his sagacity. Did he fail to diagnose the occurrence, his aim would be to recover the patient from the first shock of the accident, control excited arterial action, and relieve symptoms as they arose. And it does not appear that art could do more than this, were the anatomical nature of the affection understood from the first.

VARICOSE ANEURISMS OF THE ARCH OF THE AORTA.

I.—COMMUNICATION WITH THE SUPERIOR VENA CAVA.

1552. A woman, aged fifty, having suffered for some years from aneurismal symptoms, one evening while stooping at laborious work, suddenly felt as if strangled, changed colour, felt giddy, and sat up all night in dread of suffocation. When seen the next day, the face and upper part of the body were deeply cyanosed, the tributaries of the upper cava enlarged, those of the lower natural. The ordinary signs of aneurism of the arch existed about the right infra-clavicular region. Dr. Mayne, the observer of the case, arguing from the existence of a powerful superficial whirring systolic murmur, loudest at the second right cartilage, accompanied with thrill, perceptible not only at the spot, but, in spite of great œdema and swelling, over the right internal jugular and subclavian veins, and taking into consideration the extreme suddenness with which the serious symptoms above referred to set in, made the diagnosis of an aortic aneurism communicating with the superior cava. The pulse full, strong, and jerking, beat about 110 in the minute.

Death ensuing on the tenth day from the patient's sudden seizure, an opening "in size and shape resembling the button hole of a shirt, and crossed about the centre by a delicate frenum," was found between the vena cava and an enormous globular dilatation of the arch.* The heart exhibited nothing abnormal: the jerking pulse, it may therefore be presumed, depended directly on the loss of arterial current, sustained through escape of blood from the sac into the vein.

II.—COMMUNICATION WITH THE PULMONARY ARTERY.

1553. If an individual, known or not known to have been the subject of aortic aneurism, suddenly experience after effort a sensation of something giving way in the cardiac region, feel faint, become pale and exhibit the general characters of nervous shock to the heart, followed by peculiar fluttering in the chest,—if he subsequently suffer from dyspnœa to orthopnœa, blueness of the lips, pallor of the face, chilliness, prostration of strength, anxiety, terrible dreams, occasional nausea and vomiting, syncopal and pseudo-epileptic attacks, and become anasarcous in the lower extremities, while the lungs and liver undergo mechanical engorgement, as proved by percussion,—if all this co-exist with powerful systolic thrill, limited to the second and third left interspaces close to the sternum, and loud whirring murmur essentially systolic and intermittent [524], though sometimes inclining to continuousness, the diagnosis of communication between the aorta and either the pulmonary artery or right ventricle is warranted. The further determination of the existence or not of aortic dilatation must turn on its own special signs.

1554. Information is wanting as to the possible duration of life after such communication has been accomplished. A case recorded by Dr. Smith, seems to show that existence may be prolonged for three months; here the patient died in a pseudo-epileptic fit.* I have before me a preparation in which the vessels communicate by an opening, round, smooth, and half an inch in diameter on the aortic side, round, of the size of a split pea on the pulmonary:† according to the catalogue, the existence of the aperture was "not detected during life by any morbid sound or symptom."

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* Dublin Hospital Gazette, Feb. 1854.

III.—COMMUNICATION WITH THE RIGHT VENTRICLE.

1555. A man, aged twenty-five, while in his ordinary health, felt a "crack in the heart," and became faint and pale, while lifting a sack of flour. Though very ill, he continued at work for three or four days. Nine weeks later, Dr. Hope found the face bloated and purplish, the legs very, the hands slightly, œdematous, the pulse 80, and excessively jerking; no pain; the least effort causing dyspnœa and irregularity of pulse for two or three minutes: and the following physical signs existed,—thrill in the third interspace‡ two inches from the sternum, with very loud superficial sawing murmur, like a whispered *r* at the same spot, most marked in systole, less in diastole, with also a continuous rumbling sound,—no thrill nor murmur above the clavicles, impulse of the heart not materially increased. The patient died, highly anasarcous, nine weeks after the examination. A sac, as large as a small hen's egg, immediately above the aortic valves, opened by two small apertures into the right ventricle.

In a male, aged thirty-one, observed by Mr. Beck, both sounds are represented to have been distinctly audible; and immediately following, and loudest just after the second sound, a sawing murmur disagreeably close to the ear, accompanied with thrill, most distinct at the base of the heart near to the sternum,—but at which side of that bone is not stated,—was heard.§

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* Dublin Journal, vol. xviii.

† U. C. Museum, No. 2254.

‡ Although this is not stated in the narrative, the left interspace is evidently meant.

§ Med. Chir. Trans., vol. xxv.

IV.—COMMUNICATION WITH THE RIGHT AURICLE.

1556. There is reason to believe that where communication is established with the right auricle, the attendant thrill and murmur will be of maximum strength in the second and third right interspaces.

1557. Little, if anything, is known of the duration of life subsequent to this perforation. An ostler, aged thirty, was brought in dead to University College Hospital at about half-past ten p.m.; that evening he had been heard to groan by a fellow-workman, and answered to inquiries as to the cause, that "he should soon be better;" after this, having taken some tea and a small quantity of gin, he fell in the street while returning to work at about ten p.m. and died almost immediately. An aneurism of the ascending aorta, three inches in diameter, had opened by a rupture, jagged on its aneurismal, smooth on its auricular surface, into the right auricle,—when opened out, the communication seemed about as large as a fourpenny piece.*

This man had been known to have suffered for a long time from pain in the chest, increased by stooping; still he was looked upon by those who knew him as a healthy person: the aneurism alone did not seriously disturb him. The rupture was probably partially effected at the time of the groan, completed at the moment of the fall.

DISEASES OF THE PULMONARY ARTERY.

INFLAMMATION OF THE PULMONARY ARTERY.

1558. In rare instances of acute rheumatism, in pyohæmia, ınd various other disordered blood-conditions, in phlebitis, ıterine and other, in Bright's disease, in certain forms of ɔneumonia, and lastly, as an idiopathic state, inflammation of the pulmonary artery has occasionally been observed. But unfortunately narratives supply only scanty and loose clinical details; the disease does not seem ever to have been diagnosticated. The importance of the complaint, when it exists, can probably be with difficulty exaggerated; the basis of future investigation is admirably traced out in Dr. Chever's Essays.*

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* U. C. Museum, No. 4026, described by Mr. St. John Edwards, "Medical Times," Dec. 1851.

RUPTURE OF THE PULMONARY ARTERY.

1559. A few cases of idiopathic and traumatic rupture of this vessel are on record, but without clinical histories. In all sudden death occurred.

RUPTURE OF THE RIGHT PULMONARY ARTERY.

1560. A man, aged sixty-three, long the subject of bronchitis, the signs of which were alone discovered by auscultation, had a sudden attack of violent hæmoptysis. The bleeding, at first controlled, recurred, and death followed from exhaustion on the fourth day; the right pulmonary artery had undergone rupture just at its entrance into the lung; there were no tubercles or other new product in the tissue of the lungs.†

GENERAL DILATATION.

1561. General moderate dilatation of the pulmonary artery, trunk, and branches, has, in one or two instances, fallen under my notice, in connection with dilated hypertrophy of the right ventricle, sequential to long standing vesicular emphysema. But I do not know of any signs or symptoms whereby it might be diagnosticated. Skoda speaks of uniform widening of the vessel as a frequent state, but has not known it carried to sufficient amount to deaden the percussion-sound. Dr. Stokes gives the prominent features of a case where the dilatation was sufficient to render the valves incomplete,—but there were unluckily so many other associated morbid states in the heart and aorta, that the narrative practically contributes nothing to future diagnosis.

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* Medical Gazette, 1846—51.

† Fearn, Provincial Journal, 1845.

SIMPLE OR COMPOUND SACCULATED ANEURISM.

1562. Sacculated aneurism of the pulmonary artery, a very rare disease, may dilate the vessel to the size of a goose-egg and upwards; it may be simple or compound (Diag. III., Figs. 3 and 4); and, as in a case observed by Skoda, accompanied with extreme narrowing of both the main branches; the valves may be natural or contracted. The aorta has been sound, slightly dilated or first dilated and then contracted near the ductus arteriosus; the left ventricle may be natural; the right has been dilated and hypertrophous. In one case the ventricles communicated by a permanent aperture.* Bronchitis, pneumonia, and hydrothorax seem sequential states.

1563. The physical signs in indubitable cases have somewhat varied. The most satisfactory, actually observed, have been pulsating prominence, centralising in the second left interspace, close to the sternum, and the seat of strong systolic thrill; and systolic, superficial, loud, harsh, rasping or grating murmur, of maximum force at the prominence; and followed, in one instance, where a pulmonary valve was contracted, by a very short diastolic murmur. But in Skoda's case there is not a single sign mentioned fairly referrible to the aneurism; he himself supposes the absence of murmur explicable by the narrowness of the two main divisions of the vessel.

1564. The prominent symptomatic states have been lividity of face, described as of cyanotic depth in some cases, anasarca, ascites, great dyspnœa, cough and frequently recurring bronchitis, scanty urine, suppression of the catamenia, cephalalgia, and pain in the chest or at the epigastrium. The pulse, of variable frequency, is without special character.

But the most apparently significant symptom of the series may be wanting: Dr. Fletcher's patient was pale instead of being livid, though the two ventricles communicated besides.

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* Fletcher, Med. Chir. Trans. vol. xxv.

1565. Pulmonary aneurism has been observed in both sexes; in persons in the prime of life and in girls under twenty.

1566. Information is yet wanting as to the duration of life. In Skoda's case death followed the appearance of dropsy in three months; Dr. Fletcher's patient lived certainly twenty months, probably three years, with the disease.

1567. The diagnosis of aneurism of this vessel may be considered tolerably certain, if pulsating prominence limited to the left of the sternum and the second and third cartilages, furnishing strong, superficial, systolic, thrill and harsh murmur of the same rhythm—neither of them discoverable at the top of the sternum or above the clavicles—coexist with deep lividity of the face and anasarca, for which symptoms no ordinary explanation can be found. But aneurism of the ascending and transverse part of the arch of the aorta may protrude mainly to the left of the sternum [1501]; and tumor in the mediastinum, pressing on, and receiving impulse from, the pulmonary artery, as also great hypertrophy of the left auricle, may simulate more or less closely these states; lastly, pulsating empyema must not be altogether forgotten.

1567.* An example of dissecting aneurism of the vessel, apparently fatal in about twenty-four hours, has been recorded by Dr. Helmbrecht.*

ANEURISM OF THE DUCTUS ARTERIOSUS.

1568. A male infant died on the third day of its existence with lividity of face, difficult respiration, stifled cry, small, weak frequent pulse, but natural temperature of the surface; the ductus arteriosus dilated to the size, and in the form of a cherry-stone, was almost filled with clots; a passage, sufficient to admit a crow-quill remaining pervious.†

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* Caspar's Wochenscrift, 1842.

† Billard, Maladies des Enfans Nouveaux-nés.

APPENDIX.

CHANGE OF CLIMATE.

1569. Although we cannot at the present day subscribe unconditionally to the apophthgm "*pessimum* ægro cœlum est, quod ægrum fecit," experience justifies us in placing among the most important agencies, for modifying the course of various chronic diseases, migration from one climate to another; and, further, experience likewise proves that among chronic maladies so remediable, those of the lungs and circulating organs hold the highest rank.

1570. In changing climate we change temperature, absolute and relative from day to day and month to month, moisture, atmospheric pressure, electrical states, force and steadiness of insolation, amount of horizontal movement of the air, and certain other physical conditions. In these points of view the subject of change of climate is amply and profoundly illustrated in the classical volume of Sir James Clark,—a work which, as Isensee justly observes, " creates an epoch " in the progress of medical geography and therapeutics.*

1571. But there are influences of climate beyond the range of meteorological investigation,—climates, in truth, of closely the same atmospheric characters produce effects very dissimilar on the animal economy. We cannot, in other terms, announce *à priori* the influence which any one climate will exercise upon the inhabitant of another, though the meteorology of both regions be perfectly worked out according to the standard of existing physics. The physiological influence of all varieties of change of climate must be observed to be understood; and *à fortiori* the influence of such changes in morbid states of the frame. Now a series of precise observations on invalids is yet wanting; and until this want be supplied, attempts at fixing the climate fittest for any particular form of disease must, it appears to me, result in frequent disappointment.

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* Geschichte der Medicin, p. 1585, Berlin, 1844.

1572. Still some general truths have been acquired on the pathological branch of the subject; and one of the most important seems to be, that the anatomical condition and the presumed intimate nature of any affection are less faithful guides, than the state of the organism generally and the liking of the individual, in the selection of a dry and bracing or a moist and relaxing climate.

1573. In order to facilitate the selection of a winter place of abode for pulmonary and cardiac invalids, the following classification of climates is offered,—with the previso, however, that it is only in point of general character the different spots brought together can be held to agree. The qualities ascribed to each group are supposed to be most marked in the order the various places are mentioned.

1574. I. *Climates distinguished by soft and relaxing character, combined with moderately high thermometeric range.*—The Azores,* Rome; Pisa; Pau; Cove; Torquay; Penzance.† These climates, as a general rule, agree with pulmonary and cardiac affections, attended with dry bronchitis and a dry irritable state of skin, especially if the invalid be constitutionally but little disposed to suffer from general langour.

1575. II. *Climates less moist and relaxing, of somewhat higher thermometeric range, and slightly more stimulant than those of Group I.*—Madeira; Teneriffe;‡ Palermo. Either of the two islands (the former is more relaxing than the latter) will be appropriate for persons in whom there is no prominent demand for a relaxing or bracing, but simply for a steadily warm and genial, air: Funchal, from its superiority in the comforts and appliances of civilised life, will probably long be preferred to Santa Cruz. Palermo supplies a transition to the next group.

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* Bullar, "A winter in the Azores."

† Sir John Forbes, "Med. Topography of the Land's End."

‡ Pinkerton, Climate of Teneriffe; Monthly Journal, 1854.

1576. III. *Climates essentially dry and hot, with keen stimulant quality more or less predominant.*—Malaga; Nice; Malta; Algiers; Cadiz; and San Lucar. This group of climates is well adapted for cases in which relaxation is at once a notable character of the existing disease, and of the constitution at large. Cases of chronic bronchitis, with copious discharge; of phthisis, similarly characterised; of feeble dilated heart; of humid asthma and allied affections, benefit strikingly by the air of these places. A dry, irritable state of mucous membrane, and chronic laryngo-pharyngitis of whatever type, are made worse by them all. These spots, too, especially Nice and Malta, exercise a peculiarly exciting effect on the nervous system of some persons.

1577. IV. *Climates possessing in a less degree the essential attributes of Group III.*—Hyères; Cimiez (Nice); Villa Franca; Cannes.

1578. V. *Climates distinguished by warmth, dryness, and steadiness of temperature, without notably stimulant or relaxing quality.*—The Nile; Rhodes. The most remarkable effects I have observed in regard of the apparent suspension of phthisis, through the influence of climate, have been the results of life in a Nile-boat; yet even to Egypt, the land of stilly enchantment, intellectual, emotional, and corporeal, the invalid must not repair without knowing that his courage may be rudely tested from time to time by unusual conditions of atmosphere,* an observation which will, however, more strongly apply to every other spot mentioned.

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* Thus on the occasional violence of the winds, and severity of the cold, hear the testimony of an American traveller, not the less trustworthy, because its terms are florid. "Still? why the wild winds pace up and down he Valley of the Nile, like his mad hounds howling for Actæon; like all the

1579. VI. *Places protected, more or less completely from northerly and easterly winds; and, therefore, superior as winter-residences to spots of about the same temperature, but more exposed.*—Undercliff; Hastings; Hot-wells (Clifton); Dover; Bournemouth.

TREATMENT BY THE COMPRESSED-AIR BATH.

1580. Upwards of sixty years ago the Royal Society of Sciences at Haarlem proposed, as a subject for competition, the influence of condensed air on animal and vegetable life. The queries put by the Society appear to have met with no response. At a later period Sir John Sinclair suggested, on the ground of experiments made by other persons on the lower animals, that individuals in certain states of disease, might be made to breathe with advantage compressed air. But to M. Junod we are indebted for the first authentic experiments on the human subject in the state of health.* Subsequently M.M. Pravaz,† Tabarié, Milliet, and others of Lyons have largely applied condensed air in the treatment of a variety of affections.

The apparatuses in use at Lyons under the direction of M. Milliet, are hollow spheroids of wrought iron, of various sizes, to hold one, two, or ten or a dozen persons at once. The air is pumped in by steam-engines,—and a contrivance adapted by

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ghosts of all the three hundred dynasties anterior to history, demanding to live again. . . . . Warm? why, the Howadji sat more voluminously swathed in coats, cloaks, and shawls, than mummies in their spiced bandages. They began, bravely, with sitting in front of the cabin; and warmly wrapped in winter-clothes, and only a little chilly, played that it was summer, and conversed in a feeble, poetic way of the Egyptian climate. Gradually they retreated to the divans in the cabin, and cursed the cold."—"Nile-Notes of a Howadji," by Curtis.

* Académie des Sciences, 1834.

† Emploi Médical de l'air comprimé, 1850.

which that vitiated by respiration is removed. The least pressure employed medically is about half an atmosphere, the greatest two-thirds of an atmosphere. Each sitting lasts two hours; the first half hour is occupied in increasing, the last in lowering, the pressure; an hour is consequently passed under the full excess of pressure. The mean number of baths required for various chronic diseases is said to vary from thirty to forty.

Physiologically these baths slacken the pulse and respiration as a rule; drive the blood from the surface; excite the appetite; and throw into the system an excess of oxygen. The venous circulation is stated to be rendered more active, and secretion and absorption promoted.

The pulmonary affections said to be benefitted by this treatment are, especially, acute and chronic bronchitis, asthma both humid and dry, and emphysema; and I have certainly received strong independent testimony to the fact, that relief may be obtained in the latter affection. M. Pravaz records some few cases where amelioration appears to have been effected in the first, and even the second stage of phthisis. But a much more extensive experience, than any hitherto obtained, is required on the entire subject.

Vide page 30.

Friction-fremitus.—Friction-fremitus may remain for a long time perceptible to the patient himself. John Smith (U. C. H., Males, vol. x., p. 68) first perceived it January 13th, and felt it distinctly, on full inspiration, when discharged at the close of March.

Vide page 230.

Reduplications of heart-sounds. — Here is another variety of reduplication, recently observed: first sound doubled at the left apex, single at the base; two days later the doubling inaudible at the base and at the left apex, but distinct at the right apex, or upper edge of the ensiform cartilage (Lenton, U. C. H., Males, vol. x. p. 86). And here another variety: "at left apex second sound steadily reduplicate beat after beat; at right apex this doubling less distinct; at the base the second sound is either single or imperfectly reduplicate,—the difference in the reduplication here and at the left apex is very striking; at aortic cartilage two single sounds; at pulmonary cartilage first sound full, second slightly reduplicate and murmurish." (Couch, U. C. H., Females, vol. vii., p. 343.) My experience does not tend to connect reduplications with rheumatic fever, with or without distinct endocarditis.

Vide page 359.

Peculiar physical signs during absorption of pleuritic fluid.—The patient referred to died December 29,—that is about five weeks after the percussion-sound had become pulmonary, in the main, about the inferior angle of the left scapula. "The lower lobe of the left lung so closely adherent in the corner of the pleural sac, that the upper lobe is nearly torn away from it, in the attempt to remove the whole ; bronchi large, dilated even in some parts; tissue, especially of the posterior part of the organ, firm, hepatized-loooking, instantly sinks in water without pressure; no tubercle or gray granulation." (Fosbury, U. C. H., Males, vol. x. p. 132.) Was there air in the pleural sac when the tubular percussion and pectoriloquous echo were present?

Vide page 430.

Convalescence from pneumonia.—I have seen a few cases in which firm anasarca of a lower extremity has occurred during convalescence, evidently depending on local venous obstruction, by coagulation of the blood, and sometimes with, more commonly without, imperfect evidences of phlebitis. Time, careful bandaging, saline douches, dry friction, and shampooing are the means by which the swelling is removed. It appears to me infinitely probable that the paralysis described by M. Macario [790] may have depended on slight venous obstruction in the brain, produced by coagulation.

Vide page 458.

Gangrene of the lung.—Professor Skoda has treated this affection of late in the following manner: the essence of turpentine is poured upon boiling water, and the patient directed to inhale the vapour for fifteen minutes every two hours,—sulphate of quinine is at the same time administered. Two cases of recovery are given. Wiener Zeitschrift, 1853.

Vide page 533.

Effects of cod liver oil.—Instead of producing diarrhœa, cod liver oil actually brings on constipation in a small number of persons.

Vide page 535.

Influence of stays in tuberculising the lungs.—Although it is essential that females, already tuberculised, allow as free play as possible to the lungs, I do not by any means accede to the doctrine, that the use of stays will produce pulmonary consumption. Whether this article of dress shall or shall not inflict mischief on the lungs will probably, as I have elsewhere said, "altogether depend on the amount of constriction. If this be simply sufficient to transfer the maximum chest-play from the base to the apex of the thorax (or, rather, to magnify somewhat the breathing-difference superiorly and inferiorly natural to the female), I cannot very clearly descry what evil is to come to the lungs, especially if the stays be cut bias, and be formed of yielding material. If, on the other hand, rigid wood-work or metal plates be used to stiffen stays, of which the main material is hard and cut straight, then it is conceivable, *à priori*, that serious evil may come to the lungs. Remember, however, the wide difference in the statical and dynamic mechanism of the thorax and abdomen, and you will feel at once that the fact of serious compression of the liver being produced by tight lacing, gives no shadow of proof that the pulmonary organs must suffer to similar amount, or even in similar fashion. I know not, as matter of clinical experience, what the mode of disturbance is which constriction of the base of the chest actually and demonstrably entails on pulmonary action or pulmonary structure. Still such ignorance as this is not commonly avowed; on the contrary, the mass of information on the point is held to be positive and of ominous, most ominous quality. Dr. Copland, for instance, writes, in a recent and otherwise admirable article, that the use of stiff stays produces 'ultimately a morbid state of the blood, *tubercular deposition, especially in the lungs*, hæmoptysis, anæmia, etc.' But, it may be fearlessly asserted, no single proof exists that the abuse of stays produces the specific disease tubercle. Mr. Farr, it is true, speaks thus: —'Thirty-one thousand and ninety English women died in one year of the incurable malady, consumption. Will not this impressive fact induce persons of rank and influence to set their countrywomen right in the article of dress, and lead them to abandon a practice which disfigures the body, strangles the chest, produces nervous or other disorders, and *has an unquestionable tendency to implant an incurable hectic malady in the frame*.'* But Mr. Farr omits to compare the relative mortality of the sexes in elucidating this question. Look at this table giving the mortality from consumption in three years to a million living of each sex in England and Wales:—

YEARS.	Deaths from Phthisis to 1,000,000 living of each Sex.	
	Males.	Females.
1837	3,771	4,155
1838	3,783	4,077
1839	3,722	4,015

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* Letter to the Registrar General. 1840. P. 73.

"What evidence does this table give of the dependence of tuberculisation on stays? It simply shows that the phthisical mortality of females is somewhere about 300 per 1,000,000 living greater than that of males. Granting that the female excess is really due to stays, does its amount justify Mr. Farr's strong phrases? I think not. But let me assure you, no particle of evidence exists that the moderate excess of female destruction is really traceable to the abuse of stays. Not a few arguments might be adduced, tending to prove their absolute innocence. Thus in France, as is well known, females rarely use stays until the afternoon; in England, women tighten themselves up the moment they rise in the morning; yet the excess of female phthisical mortality over the male is greater in France than in this country. Again, in certain parts of Europe, the men tighten themselves at the base of the chest, so as to produce a tolerably fair image of the figure of a wasp, and yet they do not seem thereby to increase their relative quota of phthisical mortality. Further, it will be conceded, that tight-lacing is, as a rule, pushed to greater lengths among metropolitan than among rural female populations; so that, if the influence assigned to stays be other than a figment of the brain, the plus destruction of women over males ought to be relatively greater in London than in the country. Now, such evidence as I can get at tells in precisely the contrary direction. Thus, examine these figures:*

KENT COUNTY.

	Males.	Females.
Population in 1841	232,228	236,885
Absolute deaths from phthisis	726	778
Deaths from phthisis per 1,000,000 living . .	3126	3242

METROPOLIS.

Year.	Males.	Females.
Population in 1838	913,077	971,767
Absolute deaths from phthisis	4,057	3,630
Deaths from phthisis for 1,000,000 living . .	4,443	3,735

So that, actually, where, by fair inference, the amount of stay-constriction is greatest, and its prevalence widest, (in the Metropolis), females are destroyed by phthisis to a less degree than males; whereas, amid a country population, which we may honestly assume to undergo a less mean amount of tightening, females die consumptive in notably larger proportion than males.

"No, gentlemen, if the abuse of stays produces consumption, its power to do so most indubitably remains to be proved; and while the laws of an enlightened pathology point to the excessive improbability of an essentially diathetic disease springing from a mechanical cause, I entreat you not to adopt the popular creed, that 'stays cause consumption,' unless on direct and unimpeachable logical evidence. There is quite enough in the demonstrable evils entailed by tight-lacing to justify you in warring against the abuse; you have no need to support your arguments by the unfair appeal to an imaginary mischief."

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* Vide Regist. Rep., vol. vii.

Vide page 593.

Head low in extreme cases of hydro-pericarditis.—Numerous examples, in addition to that referred to in the text, have fallen under my notice, showing that this is the habitual posture, when the quantity of fluid has passed a certain amount, the precise amount varying probably in different persons. What is the cause? It seems most likely that the posture is assumed simply because the circulation is so obstructed by pressure on the great vessels and heart, that instinctively the patient seeks the aid of gravitation to supply the brain and lungs with blood.

Vide page 677.

Mental state in fatty heart.—The mental state, immediately preceding the syncopal attacks of this disease, is sometimes very curious. Thus a very intelligent patient, who has had a considerable number of such attacks, says; "for a minute or so before unconsciousness takes place, my memory of things just passed goes; I remember invariably, or rather my fancy conjures up, one of two or three different scenes that I have been witness of; there is no delusion, mental or optical, for I know they are not before me; but I think by shutting my eyes, I could realise them."

Vide page 714.

Treatment of cyanosis.—According to the testimony of Gintrac, alcoholic fluids produce drunkenness, with unusual rapidity in, and are otherwise very injurious to, cyanotic persons. This is a point of extreme interest in reference to the doctrines of Liebig, concerning the chemistry of respiration and of phthisis.

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